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FOREWORD.

THE London School of Tropical Medicine was founded to teach and in other ways to advance the study and practice of tropical medicine. It was opened on the 2nd of October, 1899. From that date to the present its work has been carried on without interruption. Every year there have been three teaching sessions of three months each, and a variety of researches in tropical pathology have been carried out.

The original buildings were planned on a humble scale and at first the attendance of students was comparatively small. In a short time, however, as the attendance of students rapidly increased, it became evident that the School supplied a much-felt want. Concurrently, and in consequence of the recent phenomenal development of tropical medicine, the scope of the teaching had to be extended. To meet this state of affairs the general teaching laboratory had to be enlarged on three occasions, so that it is now three times its original dimensions, and special laboratories with special teachers for such subjects as general medical zoology, helminthology, and protozoology had to be instituted. At the same time the museums and library had to undergo a corresponding extension. And, further, as it is no longer possible to cover adequately the entire field of tropical medicine in a short course of three months, a system of advanced classes had to be inaugurated and additional laboratory accommodation provided where such studies or original researches could be carried on.

There is now, therefore, accommodation for 52 in the general laboratory and for 30 students in the laboratories of the teachers of special subjects, besides 8 benches for original workers.

Since the commencement of the School, altogether 1238 post-graduate students have passed through the various courses. At the time of writing there are 48 students taking the ordinary course and 10 taking the advanced or special courses.

Thus the London School of Tropical Medicine from small beginnings has become, by a process of gradual evolution, an institution of considerable magnitude and of no inconsiderable importance to the State. In the opinion of the School Committee the time has come for a further step in development. Arrangements have been made for the publication of a Journal which shall be devoted to the subject of tropical medicine and conducted by the staff together with the past and present students of the School. Such an enterprise has been urged on the Committee many times in recent years, but considerations of finance and the desire not to add further to the superabundant serial medical literature have restrained it. Lately, however, the demand for such a journal has become so insistent, and as the interests of the school seem to demand it, and as it seems to be a natural step in the evolution of the institution, the Committee has resolved to comply with what, so far as the alumni of the School are concerned, seems to be a general wish.

It is proposed as a commencement to make one issue of the Journal about the middle of each session—that is, three numbers a year, and to limit each number to about one hundred pages. Additional matter of importance, if too long for publication in the body of the Journal, such as special reports by commissioners of the School, will appear as a supplement or supplements. The various articles will be arranged under the headings of Clinical Medicine, Pathology, Bacteriology and Hygiene, General Medical Zoology, Protozoology, and Helminthology, and the teachers of these several departments will take charge of their respective sections subject to such editing as considerations of space and illustration may make necessary. This arrangement may be altered in the future as experience may indicate.

Owing to the absence of local libraries and the difficulty and expense in procuring current literature, it is often impossible for the medical man in the tropics to keep abreast of the times, even in the comparatively limited field of tropical medicine. An effort will be made in the Journal to meet this by summaries of recent important papers, particularly of such papers as do not appear as original articles or as effective summaries in the weekly medical journals published in London.

The School Committee trust that this new departure will receive the support, both as subscribers and as contributors, of all students of the School, whether old or new. At first there are bound to be difficulties and blemishes, but given the support that is confidently looked for, it is hoped that in a short time the Journal of the London School of Tropical Medicine will be worthy of the School and of use to the readers, as well as contribute to the advance of tropical medicine.

PATRICK MANSON.

I.—SOME RECENT PUBLICATIONS RELATING TO MEDICAL ENTOMOLOGY.

By Colonel A. ALCOCK, C.I.E., F.R.S., I.M.S. (Retired).

[N.B.—The term "Entomology" is here employed in its old sense, which includes all *Arthropoda*.]

1. **Handbook of the Tsetse-flies.** By E. E. Austen. Published by order of the Trustees of the British Museum. 1911.

This book is a model of what a handbook should be, all that is at present known about tsetse-flies, on the zoological side, being succinctly stated, and all the gaps in our knowledge being pointed out for the guidance of observers in the field. There are ten beautiful coloured plates by Terzi, and the few rare species that do not appear in the plates are sufficiently illustrated by text-figures.

Mr. Austen recognizes fourteen species of *Glossina*, namely, *G. palpalis*, Robineau Desvoidy, *G. caliginea*, Austen, *G. pallicera*, Bigot, *G. tachinoides*, Westwood, *G. morsitans*, Westwood, *G. longipalpis*, Wiedemann, *G. pallidipes*, Austen, *G. fusca*, Walker, *G. fuscipleuris*, Austen, *G. nigrofusca*, Newstead, *G. tabaniformis*, Westwood, *G. longipennis*, Corti, *G. brevipalpis*, Newstead, and *G. medicorum*, Austen, of which two, namely *G. fuscipleuris* and *G. medicorum*, are new to the list. *G. fuscipleuris*, which unfortunately is based on a single male specimen from the north-eastern part of the Congo Free State, resembles *G. fusca*, Walker, but differs in the colour of the pleura and in the colour and texture of the hind coxæ; *G. medicorum*, which, so far, has been found only in West Africa, resembles *G. brevipalpis*, Newstead, but is distinguished by the uniformly coloured wings, and by the structure of the male hypopygium.

The author quotes Newstead's description of *G. fuscipes* (founded on a single specimen from Uganda), but does not express any opinion upon it; he considers Newstead's *G. maculata* (also based on a single specimen) to be an unnatural specimen of *G. palpalis*; and he regards Newstead's *submorsitans* as a mere form or "race" of *G. morsitans*.

Each of the 14 species admitted by the author is critically described and figured, its geographical distribution is exactly recorded, and its binomics—so far as they have been observed—are discussed.

In addition to the critical and concise descriptions there are dichotomous synoptic tables of species, which are constructed with the author's usual discernment. In these tables the species are first split into four groups—a *palpalis* group, a *morsitans* group, a *fusca* group, and a *brevipalpis* group—each of which is independently synthesized.

The extremely moderate cost of the book marks, it may be hoped, a happy revolution in Museum financial policy.

2. **A Revision of the Tsetse-flies, based on a study of the male genital armature.** By R. Newstead : in the 'Bulletin of Entomological Research' for May 1911 : see also the same 'Bulletin' for July 1911.

This novel and stimulating paper is more likely to interest the zoologist than the medical officer. The author has examined the hypopygium of the male of all the species of *Glossina* except *fuscipleuris* and "*maculata*," and finds, among other things, that three types of claspers exist among them :—(*a*) In *G. fusca* and the other large tsetse-flies (namely *nigrofusca, brevipalpis, longipennis, tabaniformis*, and *medicorum*) the great claspers of the male are not connected by any membrane, are claw-like, and (except in *longipennis*) have bicuspidate tips ; (*b*) in *G. palpalis, caliginea, tachinoides, pallicera,* and *fuscipes* these organs are connected by a broad membranous curtain and are more or less claw-like with (except in *pallicera*) abruptly acuminate tips; (*c*) in *G. morsitans*, "*submorsitans*," *pallidipes*, and *longipalpis* the organs in question are more or less spathulate, also being connected by membrane. So far the characters of the claspers in the main confirm Austen's grouping of the species by more readily discernible external characters that are common to both sexes. Beyond this the author claims that the exact shape, not only of the claspers, but also of certain other parts of the male hypopygium, has a fixed specific value. The reviewer has examined a large series of males of one species (*G. tachinoides*) collected by one man in one locality, and finds that whereas the large claspers are remarkably constant in form, the complicated folding of the other parts of the hypopygium is hardly capable of being specifically defined in *G. tachinoides*.

3. **Further Researches on the Development of** *Trypanosoma gambiense* **in** *Glossina palpalis*. By Colonel Sir David Bruce and the Sleeping Sickness Commission of the Royal Society ; in 'Proceedings of the Royal Society,' No. B 567, May 1911, p. 513. The authors summarize the experimental data for the following conclusions :—

Trypanosoma gambiense is never found in the proboscis of *G. palpalis* except immediately after an infected feed.

A few days after an infective feed the trypanosomes disappear out of the great majority of the flies, but in a small percentage this initial disappearance is followed by a renewed development.

After a very short time the flies which have fed on an infected animal become incapable of conveying the infection by their bites, and this incapacity lasts for some 28 days, when a renewed or late infectivity takes place.

A fly in which this renewed or late infectivity occurs can remain infective for at least 96 days.

An invasion of the salivary glands occurs at the same time as this renewal of infectivity, and without this invasion of the salivary glands there can be no infectivity.

The type of Trypanosome found in the salivary glands when the fly becomes infective is similar to the short stumpy form found in vertebrate blood, and it is believed that this reversion to the blood-type is a *sine quâ non* in the infective process.

4. **Sleeping Sickness Bureau Bulletin**, No. 29, August 1911, p. 241. The Editor states that he has been informed, through Dr. Kleine, that Dr. Taute in Tanganyika has succeeded in transmitting "a human trypanosome" to monkeys by means of *Glossina morsitans*.

5. **Remarks on the Classification of the Culicidæ, with particular reference to the Composition of the Genus *Anopheles*.** By A. Alcock: in the 'Annals and Magazine of Natural History' for August 1911, p. 240.

The author upholds the old arrangement of the *Culicidæ* in two subfamilies —*Corethrinæ* and *Culicinæ*, and divides the *Culicinæ* into four sections, namely (1) *Megalorrhini* (=*Megarhininæ*, etc. of Theobald), (2) *Epialurgi* (=*Anophelinæ* of Theobald), (3) *Culicales* (=*Culicinæ*, *Heptaphlebomyinæ*, *Dinoceratinæ*, *Aedinæ*, *Uranotæniinæ* of Theobald), and (4) *Metanototricha* (=*Dendromyinæ*, etc. of Theobald), and regards the last of them as a quite conventional group.

He gives reasons for recognizing only one genus of *Epialurgi*, namely *Anopheles*, which he divides into subgenera and "series" as follows :—

(*a*) Subgenus *Anopheles,* in which the vestiture of the scutum consists mainly of hairs, the abdomen has no scales (very rarely a few inconspicuous scales may be present on the genital lobes), and the wings are either unspotted, or have a few dark spots formed by clumps of scales, or may have a few colour spots, in which case not more than two distinctly formed spots of colour are present on the front edge of the wing. This subgenus includes *Anopheles* and *Stethomyia*, and *Cyclolepidopteron grabhamii* of Theobald's Monograph, and *Neostethopheles* and *Patagiamyia* of James.

(*b*) Subgenus *Myzorhynchus*, in which the vestiture of the scutum consists mainly of hairs : abdominal scales are restricted to a single tuft on the ventral surface of the penultimate segment except in one species ("*Lophoscelomyia*"), where the last segment and the genital lobes are covered with scales ; the wings are not much spotted, and never have more than two spots on the front edge ; the wing-scales are rather broadly elliptical ; and the palpi are shaggy. This subgenus includes *Myzorhynchus* and *Lophoscelomyia* of Theobald's Monograph.

(*c*) Subgenus *Christya*, differing from *Myzorhynchus* only in the abdomen, which, in addition to scales on the genital lobes and terminal segment, has on every segment a pair of lateral wisps of slender and extremely long scales.

(*d*) Subgenus *Arribalzagia*, restricted to Central and South America. Here the vestiture of the scutum consists mainly of hairs ; broad scales, which sometimes stand out as tufts, are present on all the segments of the abdomen: the wings have a dark cast, owing to predominance of blackish scales ; the wing-scales are broad, and the palpi are shaggy. The subgenus includes *Arribalzagia*, *Manguinhosia*, *Kerteszia*, and part of *Cyclolepidopteron* of Theobald's Monograph.

(*e*) Subgenus *Myzomyia*. Here the vestiture of the scutum consists mainly either of hairs, or of very narrow scales that may sometimes be confused with hairs ; there may be a few scales on the genital lobes and last segment of the abdomen ; and the wings are profusely spotted, the costa being barred with numerous alternate dark and light spots or streaks. In this subgenus two not very distinctly separable series of species can be recognized, namely, *Myzomyia*, in which hairs predominate on the scutum : and *Pyretophorus*, in which narrow scales predominate on the scutum. The subgenus includes *Myzomyia*, *Feltinella*, *Neomyzomyia*, and *Pyretophorus* of Theobald's Monograph, and James's *Nyssomyzomyia*.

(*f*) Subgenus *Nyssorhynchus*. Here the scutum is fairly well covered with recumbent elliptical scales of considerable breadth ; scales are almost always present on some part of the abdomen ; the wings are much speckled with numerous spots on the front edge; the palpi are shaggy; and the legs are generally profusely speckled or brindled. The subgenus includes *Nyssorhynchus*, *Neocellia*, and *Cellia* of Theobald's Monograph, *Calvertina* of Ludlow, and *Christophersia* of James. The species of *Nyssorhynchus* may be arranged in three series, namely: *Nyssorhynchus*, in which scales are restricted to the terminal part of the abdomen; *Neocellia*, in which scales are scattered on all the abdominal segments; and *Cellia*, in which, in addition to a general investment of scales, there are also outstanding tufts—lateral or ventral—on the abdomen.

(*g*) Subgenus *Chagasia*, restricted to Tropical America, and differing from *Nyssorhynchus* chiefly in the form of the head-scales, which are not "forked." The subgenus includes *Chagasia* of Cruz, and *Myzorhynchella* of Theobald.

The "genus" *Aldrichia* or *Aldrichinella* is not accepted; and *Bironella*, which is founded only on male specimens, is treated with reserve.

In the treatment of the *Culicales* the palpi, *per se*, are regarded as of subordinate importance, and the numerous species are grouped in seven series round *Culex*, *Stegomyia*, *Aedes*, *Uranotænia*, *Psorophora*, *Mucidus*, and *Acartomyia*. *Heptaphlebomyia* and *Dinocerites* are included in the *Culex* series, *Dinocerites* being regarded merely as a *Culex* adapted to a special mode of life. The author regards the *Mucidus* series as annectant between the *Epialurgi* (*Anopheles*) and the *Culicales*.

6. **The Papataci Flies** (*Phlebotomus*) **of the Maltese Islands.** By R. Newstead; in the 'Bulletin of Entomological Research' for May 1911, p. 47.

Since the discovery of the part played by the "Papataci" (*Phlebotomus papatasii*) in carrying the infection of a specific autumnal fever of Southern Europe, the genus *Phlebotomus* has attracted a good deal of attention, and this paper, which gives a fine account of the anatomy, post-embryonic development, habits, and specific characters of the forms found in Malta, will be cordially approved by medical men in the many parts of the world where the genus is represented, since Grassi's monograph 'Ricerche sui Flebotomi' is not easy to obtain.

In Italy Grassi found the larvæ of *Phlebotomus* among builders' rubbish in cellars; in Malta Marett found them in crevices in the walls of caves and in the crannies of damp masonry: Newstead had not much luck in his search for them, and says that they were difficult to detect even in places where they were known to exist. He describes and figures the larva and pupa of *P. papatasii*: the larva is characterized by the enormously long caudal bristles, of which there are two in the young and four in the full-grown larva; the pupa is recognized by the presence of the shrivelled larval skin, with the long caudal bristles attached, at the after end of the abdomen. The habits and season of the adult flies are discussed, and there is a good and well-illustrated account of the anatomy, external and internal.

7. **A Revision of the Oriental Species of** *Stomoxys*. By Sophia L. M. Summers: in the 'Annals and Magazine of Natural History' for August 1911, p. 235.

This is a critical account of the ten species of *Stomoxys* known to occur in the Oriental region, and is illustrated by 5 text-figures. The difficulties of recognizing the specific characters are pointed out by the authoress, who, however, has succeeded in drawing up a compact and precise table for the recognition of the species. It is pointed out that *Stomoxys calcitrans*, which is as cosmopolitan as the common housefly, is a very variable species—so variable that it has been described under at least twenty-one different names. Of the ten species dealt with, one, *Stomoxys pratti*, is described as new.

8. **A Monograph of the Ixodoidea.** Part II. By G. H. Nuttall and C. Warburton. Cambridge University Press.

In the magnificence of its proportions and its scrupulous attention to minutiæ this great work may be likened to the famous monuments of the Mogul Emperors. It is truly a monument of sedulous research; nothing that has ever been written about a tick seems to have been overlooked or left untested.

The present part consists very largely of an analysis of the genus *Ixodes*, of which the authors recognize 51 species as valid. All these species (and the varieties of certain of them) are fully described, every species, with the exception of only two, being adequately illustrated by text-figures; there are also elaborate keys (which, so far as is possible, include males, females, nymphs, and larvæ), and exact statements of specific and geographical occurrence.

In addition to this exhaustive account of the species of *Ixodes*, all the genera of *Ixodidæ* are individually characterized, and there are two appendices by Nuttall—one on the process of coupling in *Ornithodorus moubata*, the other on the adaptation of ticks to the habits of their hosts.

In the definition of genera *Aponomma* is treated as a subgenus of *Amblyomma*, and *Ixodes* is placed in a section by itself.

The authors discuss the classification of ticks in all its aspects, and maintain the two families *Ixodidæ* and "*Argasidæ*." The *Ixodidæ* are divided into two sections, namely *Prostriata* (for *Ixodes*), in which the arch of the anal groove is in front of the anus, and *Metastriata*, in which the anal groove, when present, arches behind the anus. The *Metastriata* again are arranged in two series according to the length of the rostrum, namely *Brevirostra* and *Longirostra*.

9. **Ixodidæ in Schulze's 'Das Tierreich.'** Lieferung 26. Berlin. 1911. By G. Neumann.

This is Neumann's latest word on a group to which he has given prolonged and profound attention, and it contains a systematic account of the whole family. In marked contrast to other recent authors, Neumann's *Ixodidæ* embraces not only all the ticks, but also the curious bat-parasite *Spelæorhynchus*, in which the chelicerae and the styliform hypostome lie in a rostral cavern, and the pedipalps are composed of 5 segments. The family as thus extended is divided into two subfamilies, namely *Ixodinæ* (equivalent to the *Ixodidæ* and "*Argasidæ*" of other writers) and *Spelæorhynchinæ*. The *Ixodinæ* again are arranged in two sections, *Ixodini* and *Argatini*, and so on.

There are keys to tribes, genera, and species, and every species accepted by the author is described, so that for the practical purpose of identifying ticks this book, by a leading authority, is exactly what is wanted at the present moment.

A. A.

II.—DEPARTMENT OF PROTOZOOLOGY.

By C. M. WENYON, M.B., B.Sc. (Lond.).

Owing to my absence abroad till the beginning of the present session I have been unable to prepare my contribution to the first number of the Journal. In future numbers, however, it is hoped that under the section devoted to Protozoology there will appear matter which will be of distinct use, not only to those who have passed through the school, but also to any medical men in the Tropics who have not access to current literature.

It is proposed in the first place to publish an account of original investigations undertaken in the Protozoological Department if the time of completion of such investigations coincides with the date of publication of the School Journal. In addition to any original contributions of this nature, a section will be devoted to reviews of the more interesting and important papers which have recently appeared dealing with Protozoological subjects, more especially those connected with the study of tropical diseases.

Former students of the School or any others at home on leave who have material collected in the tropics can, if they feel so inclined, carry out the investigation of such material in the laboratories of the Protozoological Department. Assistance will be given them both in the method of investigation and the reference to the literature relating to their subject. The School Journal will afford an excellent opportunity for the publication of the results of these investigations.

III.—DEPARTMENT OF HELMINTHOLOGY.

By R. T. LEIPER, D.Sc., M.B.

On the Morphology of *Oxyuris ambiguus*, Rud.: Type of the Genus *Passalurus*.

By A. HUTTON, M.B.,
West African Medical Staff,
late Demonstrator, London School of Tropical Medicine.

(*From the Helminthological Laboratory.*)

While engaged in studying the parasites of domesticated animals, I had occasion to look up the literature in connection with the common threadworm (*Oxyuris ambiguus*) of the rabbit, and was surprised to find that little had been added to the first brief description by Rudolphi a hundred years ago. In 1845 Dujardin created a new genus *Passalurus*, and removed thereto the *Oxyuris ambiguus* of Rudolphi. This genus was ignored, however, by Schneider and subsequent writers. It seemed to me, therefore, that it might be of interest to examine the parasite in some detail to see if Dujardin's genus was a good one, in view of its common occurrence in hares and rabbits, and as it is always a desirable thing to make a detailed examination of the type of a genus.

During the past Christmas vacation, spent in Aberdeenshire, an opportunity presented itself of examining a number of rabbits, and in every case this species of parasite occurred.

The worms live in the large intestine and cæcum, and in all the rabbits examined several hundred specimens were found. The male worms were present in greater number than the females. The sexes were easily distinguished, the males being smaller with a curled tail, while the females are longer and straight with a slight bulge on one side near the middle of the body. The tail of the female is very long and slender ; the male measures 4·5 to 5·5 mm. long, while the female is 8·5 to 10·5 mm. long. The cuticle is transversely striated, the striæ being most marked in the middle of the body. The striæ on the body are ·01 mm. apart in the male and ·0046 mm. in the female. The tail of the male is quite smooth, in the female there are striæ ·0062 mm. apart. On either side of the body is a lateral membrane which begins very gradually from the head and reaches it greatest breadth, ·025 mm., opposite the bulb of the œsophagus. The anterior part of this membrane shows six breaks at distances of ·1, ·18, ·275, ·425, ·6, and ·75 mm. from the head. The membrane is triangular in shape, measuring ·06 mm. at the base, and it does not show any striations. Towards the posterior extremity it gradually becomes less distinct and finally it ends opposite to the anus. The oral aperture is terminal and is surrounded by three lips. The mouth contains the end of the œsophagus, which is divided into three parts, each of which bears three spines, which differ in size, the largest being the most anterior and the smallest being at the bottom of the mouth. Round the mouth are six papillæ, two of them large and terminal on opposite sides of the mouth, while the other four are more posterior. The two large papillæ are of a very definite shape, being knob-shaped at the tip, while the basal part widens and then stops abruptly; they are ·015 mm. long. The œsophagus measures ·5 mm. long in the male and ·625 mm. in the female, and consists of two parts, the anterior being long and the posterior a thick muscular bulb with a narrow constriction between them. The length of the first part of the œsophagus is ·35 mm., of the constriction ·025 mm., and of the bulb ·125 mm., while these portions are respectively ·06, ·03, and ·125 mm. wide. In the female these measurements are a little greater proportionate to the greater length. The anterior part is crossed by the nerve ring at a distance of ·2 mm. from the head. The bulbous part contains first a small three-lipped valve, and next a triangular-shaped punching apparatus. At the junction of the chyle intestine and the œsophagus is another three-lipped valve larger than the first one. The chyle intestine is simple and runs backwards to end in the cloaca in the male and the rectum in the female. At first the wall is thick and much corrugated on its inner surface, but in its greater length it is thin-walled. At the junction of the chyle intestine and the cloaca are the rectal cells, three in number, very large, with a nucleus measuring ·0075 mm. in diameter; these cells are much smaller in the male. The excretory vesicle is oval in shape, situated well behind the œsophageal bulb, the excretory pore being 1 mm. from the head in the male and 1·25 mm. in the female.

Male.—Length 4·5–5·5 mm., breadth ·3 mm.; the tail is ·5 mm. in length, and is divided into two distinct parts, the first part being thick and tapering and ending abruptly in the second part; each part is ·25 mm. long. There are two large papillæ at the division between the two. The cloaca is chitinous and is ·086 mm. long. The testis begins as a solid part which lies either alongside or across the chyle intestine, and is ·25 mm. in length.

At the level of the excretory pore this becomes the vesicular part, which runs backwards with a double bend ·3 mm. long, the beginning of which is 1·7 mm. from the head. It then becomes gradually wider, and ends in the ejaculatory part by a narrow constriction. The ejaculatory portion is ·45 mm. long, and it ends in the cloaca. The spicule is dagger-shaped, ·13 mm. long and ·075 mm. broad. There is one pair of adanal and one pair of post-anal papillæ; these papillæ are large and loaf-shaped. Between the post-anals is a single unpaired papilla, the tip of which is occasionally double.

Female.—Length 8·5–10·5 mm., breadth ·5 mm.; the tail is 1·65 mm. long, and the first part of it tapers gradually to end in a tail similar in size and shape to that of the male. The cuticular striæ of the tail are wider apart than those on the body, and in some specimens the body is constricted; there are no papillæ on the tail or round the anus; the rectum is ·14 mm. long; for a distance of ·04 mm. it runs backwards in the same line as the chyle intestine and then bends sharply towards the anus. The vaginal opening is 1·6 mm. from the head in the mid-ventral line; round it are papillæ which are irregular in position, and which vary in number from two to fifteen. The vagina runs backwards, and at a distance of ·65 mm. from the opening it is frequently blocked by a plug of mucous material; it then runs backwards almost to the rectum, then bending sharply continues forwards to a distance of ·4 mm. from the vaginal opening; here it divides into the two uteri. Each uterus ends in a long irregularly coiled ovarian tubule, each of which interlaces and ends near the chyle intestine.

The eggs are of very large size, measuring ·1 mm. long and ·04 mm. broad; they are flattened on one side.

Helminthological Department,
April 1911.

CHECK-LIST OF NEW GENERA AND SPECIES OF HELMINTHES IN 1911.

BY R. T. LEIPER AND M. T. MAHER.

ACHEILOSTOMA SIMPSONI, Leiper, 1911 (n. g., n. sp., t. sp.), in a large Rodent.—N.

ANCHYLOSTOMUM BRAZILIENSE, G. de Faria, 1911 (n. sp.), in *Canis familiaris* and *Felis catus dom.*—N.

ANOMOTÆNIA RUSTICA, Neslobinsky, 1911 (n. sp.), in *Hirundo rustica.*—C.

ASTHEMIA FOXI, Goldberger & Crane, 1911 (n. sp.), in *Cebus capucinus.*—T.

AZYGIA ACUMINATA, Goldberger, 1911 (n. sp.), in *Amia calva.*—T.

AZYGIA BULBOSA, Goldberger, 1911 (n. sp.), in *Amia calva.*—T.

BERTIELLA CERCOPITHECI, Beddard, 1911 (n. sp.), in *Cercopithecis callitrichus.*—C.

CYLINDROPHARYNX BREVICAUDA, Leiper, 1911 (n. g., n. sp., t. sp.), in *Zebra.*—N.

CYLINDROPHARYNX LONGICAUDA, Leiper, 1911 (n. sp.), in *Zebra.*—N.

DASYMETRA CONFERTA, Nicoll, 1911 (n. g., n. sp., t. sp.), in *Tropidonotus rhombifer.*—T.

DILEPIS CAPRIMULGA, Neslobinsky, 1911 (n. sp.), in *Caprimulgus europæus.*—C.

DILEPIS CYPSELINA, Neslobinsky, 1911 (n. sp.), in *Cypselus apus.*—C.

DILEPIS MONEDULÆ, Neslobinsky, 1911 (n. sp.), in *Corvus monedula.*—C.

DILEPIS OCHROPODIS, Neslobinsky, 1911 (n. sp.), in *Totanus ochropus.*—C.

DIROFILARIA REPENS, Raill. & Henry, 1911 (n. g., n. sp.), in *Canis familiaris.*—N.

FILARIA BAUCHEI, Raill. & Henry, 1911 (n. sp.), in *Sus scrofa.*—N.

HAPLOCLADUS MINOR, Odhner, 1911 (n. sp.), in *Pleuronectes limanda.*—T.

HAPLOCLADUS TYPICUS, Odhner, 1911 (n. g., n. sp., t. sp.), in *Caranx trachurus.*—T.

HASSALLIUS HASSALLI, Goldberger, 1911 (n. g., n. sp., t. sp.), in *Ambloplites rupestris.*—T.

LECITHOSTAPHYLUS RETROFLEXUS (Molin), Odhner, 1911 (n. g., t. sp.), in *Belone acus.*—T.

LECHRIORCHIS VALIDUS, Nicoll, 1911 (n. sp.), in *Heterodon platyrhinus.*—T.

MICROFILARIA ROSENAUI, McCoy, 1911 (n. sp.), in *Citellus beecheyi.*—N.

OCHETOSOMA FORMOSUM, Nicoll, 1911 (n. sp.), in *Lepodera annulata.*—T.

PROCTOPHANTASTES ABYSSORUM, Odhner, 1911 (n. g., n. sp., t. sp.), in *Macrurus rupestris* and *Gadus æglefini.*—T.

PROCTŒCES ERYTHRÆUS, Odhner, 1911 (n. sp.), in *Chrysophrys bifasciata.*—T.

PROCTŒCES MACULATUS (Looss), Odhner, 1911 (n. g., t. sp.), in *Blennius ocellaris.*—T.

PROCTOTREMA BACILLIOVATUM, Odhner, 1911 (n. g., n. sp., t. sp.), in *Mullus barbatus.*—T.

RHODOTREMA OVACUTUM (Lebour), Odhner, 1911 (n. g., t. sp.), in *Drepanopsetta platessoides.*—T.

SETARIA BERNARDI, Raill. & Henry, 1911 (n. sp.), in *Sus scrofa.*—N.

SETARIA CONGOLENSIS, Raill. & Henry, 1911 (n. sp.), in *Phacochœrus porcus.*—N.

SPHYRONOCOTÆNIA UNCINATA, Ransom, 1911 (n. g., n. sp., t. sp.), in *Neotis caffra.*—C.

SPIROPTERA UGANDA, Leiper, 1911 (n. sp.), in a Monkey.—N.

STERINGOTREMA CLUTHENSIS (Nicoll), Odhner, 1911 (n. g., t. sp.), in *Pleuronectis microcephalus.*—T.

STRONGYLOIDES CEBUS, Darling, 1911 (n. sp.), in *Cebus hypoleucus.*—N.

STRONGYLOIDES NASUA, Darling, 1911 (n. sp.), in *Nasua nasica panamensis.*—N.

STYPHLODORA CONDITA, Faria, 1911 (n. sp.), in *Spilotes pullatus.*—T.

THYSANOSOMA GAMBIANUM, Beddard, 1911 (n. sp.), in *Cricetomys gambianus.*—C.

TOXASCARIS LIMBATA, Raill. & Henry, 1911 (n. sp.), in *Canis familiaris.*—N.

TRACHYPHARYNX NIGERIÆ, Leiper, 1911 (n. sp., n. g., t. sp.), in a large Rodent.—N.

ZOOGONOIDES SUBÆQUIPORUS, Odhner, 1911 (n. sp.), in *Anarrhichas lupus.*—T.

(C. = Cestode; N. = Nematode; T. = Trematode; n. g. = new genus;
n. sp. = new species; t. sp. = type species.)

THE SOURCE OF INFECTION WITH THE ASIATIC LIVER FLUKE.

[H. KOBAYASHI, 1911.—Mitteilungen aus dem Kaiserlichen Institut für Infektions-Krankheiten zu Tokio, pp. 58–62.]

IN the course of investigation of a number of molluscs, fishes, and aquatic arthropods for larval stages of trematodes, Kobayashi discovered an encysted distome in the muscle of certain freshwater fishes, especially those obtained in the villages of Kojo and Sanban near Okayama, in Japan. As these places are well known to be highly infected with *Clonorchis endemicus*, he suspected that the encysted form might prove to be the cercaria of *C. endemicus*. As cats are often found to be infected by *C. endemicus*, nine kittens and two adult cats, previously ascertained by repeated examinations of the fæces to be free from distomes, were given the flesh of fish which contained encysted distomes. During the experiments they were fed on boiled rice and disinfected milk.

Kitten No. 1 was given some flesh of *Pseudorasbora parva* on May 17th, 1910, and one month afterwards examination of fæces showed the presence of distome eggs. On dissection innumerable distomes were found in the bile-duct, gall-bladder, hepatic ducts, pancreas, and even in the duodenum.

Two other kittens (Nos. 2 & 3) were given flesh of *Leucogobio güntheri* on June 23rd. One of them was killed and examined on June 29th, and the liver was found infested with immature distomes. The other kitten died on July 3rd: the gall-bladder and bile-duct were found to be filled with immature forms of the same kind.

Lastly, flesh of *Leucogobio güntheri* and *P. parva* was given to six other kittens (Nos. 4–9), which were killed respectively after 3 hours, 15 hours, 9 days, 16 days, 22 days, 26 days; and to two adult cats, which were killed after 35 and 40 days.

The results were positive in every case, and in the last four (22, 26, 35, and 40 days) the parasites found already contained mature eggs. Further experiments were performed on several rabbits and guinea-pigs with the flesh of *P. parva*. The encysted distome is found in the whole of the muscular part and subcutaneous tissues of the fish. After several days all were found to be heavily infected. The worm when freed from its cyst measures 0·5 mm. in length by 0·1 mm. in breadth, the body tapers posteriorly. The skin is covered with very fine spines which are conspicuous in the younger stages but later disappear. The whole body is dotted with yellowish pigment. The oral and ventral suckers are distinct, the latter being situated in the anterior part of the posterior half of the body. The cyst is ovoid or elliptical in shape, measuring 0·13 by 0·1 mm. The larva constantly rotates in the cyst in which it lies with the body folded upon itself. Three hours after being fed to a cat the worms are found free from the cysts and creeping about actively; some have already reached the duodenum. After 15 hours they may be found in the gall-bladder. On the 6th day the parasite measures 1 mm. in length by 0·1 mm. in breadth; the sexual organs are not yet apparent. On the 12th day the testes, ovary, and uterus can be clearly traced, and the body is 3 mm. in length by 0·5 mm. in breadth. From 22 to 30 days after infection the uterus of the parasite becomes filled with eggs.

Mature specimens of the experimentally reared distome measure 5–12 mm. in length and 1–2·5 mm. in breadth, the size apparently depending, according to Kobayashi, upon the space and nourishment available for the parasite. In the first state the body is translucent with a slight reddish tint, the skin is smooth and without spines, and there is no pigmentation of the body. Further details are given which conclusively show that the experimentally produced parasite belongs without doubt to the genus *Clonorchis*.

Further observations by Kobayashi in other districts of Japan where hepatic distomiasis is also known to occur has revealed the presence of similar encysted forms, not only in the muscles of *Leucogobio güntheri* and *Pseudorasbora parva*, but also to a less extent in *Acheilognathus lanceolata*.

A. limbata. Paracheilognathus rhombea. Pseudoperilampus typus, Abbottina psegma. Biwia zezera, and *Sarcocheilichthys variegatus.* He thinks it very probable that this list of infected fishes will be increased on further investigation.

It should of course be noted that the fish act as the *second* intermediate hosts of the parasite. The first intermediary and the manner in which the fish become infected still require investigation. R. T. L.

STRONGYLOIDES INFECTIONS IN MAN AND ANIMALS IN THE ISTHMIAN CANAL ZONE.

[S. T. DARLING.—Journal of Experimental Medicine, vol. xiv. no. 1 (1911).]

THIS contribution contains an account of observations on the incidence of Strongyloides infection amongst Hospital and Asylum cases and the results of detailed study of the characters, in culture and in sections of tissue, of *S. stercoralis* and two new species, *S. cebus* in the white-faced organ-grinder monkey and *S. nasua* in the ant-bear. The author gives the following summary of his results :—

From a study of Strongyloides infections in man and animals in this region, it has been determined, in confirmation of the view of Grassi, Calmette, and others, that they are not causative factors in the production of diarrhœa. The mother worm, however, burrows into the mucosa and deposits her ova there. Certain tissue reactions take place and are evidenced by the cellular proliferation in those portions of the intestine occupied by the Nematodes.

In animals there is an associated anæmia, not positively attributable to the Strongyloides but, on the other hand, not attributable to any other cause. It is possible that *S. stercoralis* may cause some degree of anæmia in man. The amount is indeterminable in this region among hospital cases on account of the associated hook-worm disease or malaria.

Portals of entry for various micro-organisms are made by the female mother worm and her larvæ in the small intestine, and, while no case of general bacterial infection has been proved to have arisen in this way, its occurrence is possible and highly probable.

In the cultures of Strongyloides of man there is among natives, who presumably are infected with purely tropical strains, a very marked predominance of development by the indirect or sexually differentiated mode, in some cases an absolutely pure culture of the indirect mode larvæ being obtained. There are, however, natives, cultures from whose stools contain from a single filariform larva of the direct phase up to a very definite predominance of this mode. Cultures from natives of the temperate zones contain a marked predominance of the direct phase larvæ.

The presence of the filariform (direct mode) larva is perhaps best accounted for by its being an attempt at more perfect parasitism (Stiles).

From a correlation of culture study with the results of a histological examination of the invaded mucosa, the explanation of the derivation of the two phases is suggested. The mother worm in the intestinal tract has two kinds of progeny : (*a*) those expelled into the crypts of lumen ; and (*b*) those embedded in the intestinal wall. One lot becomes larvæ of either the direct or indirect phase, the other lot of the opposite phase. It appears to me that the intra-epithelial cell-developed larvæ furnish the direct phase, while the embryos expelled directly from the mother worm become the indirect phase larvæ.

Cold merely inhibits the development of the larvæ into either the filariform (direct) or into the sexually differentiated adults, and does not alter the anlage. The resistant as well as the infecting form is the filariform larva, and all chemical larvicides must be directed against this form. Thymol and an alkaline cresol resin soap were found to be effective larvicides for the filariform larvæ. R. T. L.

Notes of Recent and some New Records of Helminthes in Man of which there are few Records.

By ROBERT T. LEIPER, D.Sc., M.B.

Davainea madagascariensis.

Garrison[*] has recently extended the geographical distribution of this parasite by recording its discovery, in two cases, by Andrews at Manila, Philippines. Only eight infections had been previously reported, viz., one each by Davaine from the Comorro Islands; Leuckart from Bangkok, Siam; Daniels from British Guiana; Blanchard from Nossi-Bé near Madagascar; and four by Chevreau from Mauritius. Garrison adopts the suggestion of Blanchard that possibly the cockroach will be found to act as the intermediate host of the parasite.

Fasciolopsis buski.

Mathis and Leger[†] remark on the fact that whereas Noc found this parasite in 16 out of 133 natives of Cochin China, they have found it in one case only in Indo-China during a prolonged investigation, although they noted its presence in no less than 6 per cent. of the pigs slaughtered at the abattoirs of Hanoi.

Gastrodiscus hominis.

The first record of the occurrence of this parasite in man in Cochin China has just been published by Brau and Bruyant[‡]. In view of the supposed rarity of this helminth it is worthy of note that in addition to the type specimens of Lewis, the London School of Tropical Medicine has received specimens from (1) Sir Patrick Manson, (2) Dr. Wise, British Guiana, (3) Prof. Hewlett. I have also recently found a small number, from a single case, in the Cobbold Collection in the Royal College of Surgeons Museum, London. The specimens numbered 600 when collected.

Fascioletta ilocana = Echinostomum ilocanum.

Odhner[§] has recently published a paper on the anatomy and systematic position of this parasite. He demonstrates the presence of a crown of spines at the anterior end of the body, which, on account of their minuteness, had probably been overlooked by Garrison. A few of these spines can be made out in the specimen presented to the London School of Tropical Medicine by the discoverer of the parasite. The presence and arrangement of these spines, together with other characters, necessitate, as Odhner points out, the transference of the species from the new genus *Fascioletta* to the old genus *Echinostomum*.

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[*] The Philippine Journal of Science, vol. vi, no. 3 (B), June 1911.
[†] Bulletin de la Société de Pathologie Exotique, vol. iv, no. 4 (1911).
[‡] Bulletin de la Société de Pathologie Exotique, vol. iv, no. 7 (1911).
[§] Zoologischer Anzeiger, August 1911.

FILARIA VOLVULUS = ONCHOCERCA VOLVULUS.

G. M. Gray *, of the West African Medical Staff, in a paper from the Helminthological Department of the London School of Tropical Medicine, has described two new cases of *Filaria volvulus* from Southern Nigeria. This uncommon parasite had been reported previously from the Congo, Cameroons, Uganda, Dahomey, Sierra Leone, and Northern Nigeria †.

ŒSOPHAGOSTOMUM BRUMPTI = ŒSOPHAGOSTOMUM APIOSTOMUM.

This parasite was first described in 1902 from immature forms found by Brumpt in cysts in the wall of the great intestine of a native near Lake Rudolph in East Africa. Brumpt has also obtained it from various African monkeys. In April of the present year adult forms were found in a collection of Ankylostomes sent to us ‡ at the London School of Tropical Medicine by Dr. Andrew Foy, of the Northern Nigeria Sanitary Service. A careful comparison of these with specimens of *Œsophagostomum apiostomum* collected from monkeys in Uganda reveals no specific differences. Under these circumstances the name *Œ. brumpti* must give way to *Œ. apiostomum*, as the latter name is older than the former.

TRIODONTOPHORUS DEMINUTUS.

Like the preceding, this parasite occurs also in monkeys. The original specimens remained in the Museum of Paris undescribed and un-named from 1865 until 1905. Since the latter date it has been found in natives of Nyasaland §, notably by Dr. Turner, Transvaal, who has met with it in several cases (in one instance more than sixty examples were collected), by the Medical Officer of Lorenco-Marques, and still more recently by Dr. Stannus, of the Colonial Medical Service in Nyasaland.

Railliet and Henry have transferred this form from the genus *Triodontophorus* to a new genus, called *Ternidens*, on account of the terminal splitting of the two dorsal rays. The parasite is retained here in its old genus because the characters of the mouth-capsule seem of much greater systematic value than the terminations of the posterior rays. A similar splitting of the bursal rays is one of the specific characters which distinguish *Cylindropharynx brevicauda* from *C. longicauda*, two species in the zebra, which possess mouth-capsules that differ only in minute details. The artificial nature of the classification propounded by Railliet and Henry is further demonstrated by the fact that they are forced to place the genera *Ternidens* and *Triodontophorus* in separate groups with forms in which the mouth-capsules are highly dissimilar.

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* Journal of Tropical Medicine and Hygiene, April 1911.

† See Parsons, 'Parasitology,' 1909.

‡ Journal of Tropical Medicine and Hygiene, 1911.

§ Leiper and Turner, Journal of Tropical Medicine and Hygiene.

STRONGYLUS GIBSONI = S. DIGITATUS = S. FORDI = NEMATODIRUS FORDI (Daniels), mihi.

This parasite is described as having been collected by a native abattoir attendant at Hong Kong from his own fæces and presented to the Veterinary Officer in charge. It is described as a new parasite of man and as a new species by Stephens. A comparison of the original specimens with those obtained from the stomach of a calf in the Malay States reveals no specific differences. The parasite, it would appear, had already been found in Ceylon and named by v. Linstow as *Strongylus digitatus*, and Daniels had seen and recorded the same form at Kuala Lumpur even earlier under the name *Strongylus fordi.* The long filiform spicules and the absence of the dorsal ray and supporting membrane in the bursa of the male suffice to associate the species along with *Strongylus filicollis* in the genus *Nematodirus.* From what is known of the life-history of the allies of the parasite and from the fact that the patient shortly afterwards left his position at the abattoir, one cannot but feel very grave doubt regarding the manner in which the native procured the specimens.

GNATHOSTOMA SIAMENSE = G. SPINIGERUM.

Two descriptions only have appeared of this species as a parasite of man. The female was described by Levinsen in 1889, and the male by Leiper in 1908 *. In the latter paper it is suggested that this parasite is not a natural parasite of man, but is an accidental infection, with a species probably having a carnivore as its normal host.

During a recent visit to the Court Museum in Vienna the writer had the privilege of studying the original specimens of *Gnathostoma robustum*, Diesing, from the leopard, which is acceptedly the same as *Gnathostoma spinigerum*, Owen, of the tiger. There is no doubt that the male form obtained from man corresponds exactly to the male of *G. robustum*, while the description given by Levinsen tallies in every point with the females in Diesing's collection.

G. spinigerum has been found lately by S. Mitter in the cat in Calcutta, and there seems but little doubt that it will prove to be a parasite of the domesticated cat in Siam also, where alone it has been found in man up to the present time.

PHYSALOPTERA MORDENS.

This species, unless it be proved later to be identical with *P. caucasica*, appears to be confined in its distribution to Tropical Africa. The first find was made by Captain Gray, of the Royal Society Sleeping Sickness Commission in Uganda, and was recorded in 1909 by myself. Since then several cases †, in some cases with heavy infections, have been secured by Dr. Turner amongst Nyassa natives. I have recently found specimens of the same species which had been collected from "*Simia* sp.," Angola, in the Court Museum, Vienna. The latter were labelled *Physaloptera dilatata*, with the type specimens of which, however, they do not conform.

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* 'Parasitology,' vol. i. 1909.

† Leiper, Journal of Tropical Medicine and Hygiene, July 1911.

GIGANTORHYNCHUS GIGAS.

The records of the occurrence in man of this common parasite of the pig are not at all clear. Lindemann asserts that it is frequent among the inhabitants along the banks of the Volga.

For some years an occasional egg of this species has been noticed by students at the London School of Tropical Medicine in the course of their examination of a supply of egg-containing fæces which originally came from Hong Kong. In view of the great frequency of the parasite amongst Chinese pigs, as recently demonstrated by Dr. Maxwell, it is surprising that it has not been noted from time to time in man in that part of the world.

GIGANTORHYNCHUS MONILIFORMIS.

This is a common parasite of rats in various parts of the world. Magalhæs has found the encysted larvæ in the cockroach in Brazil. More recently Professor Powell, of Bombay, has sent to us specimens from the same source found in the fat-body of cockroaches in Bombay. Calandruccio by auto-infection showed that man was a facultative host, but no case of natural infection appears to have been recorded. Some months ago Dr. Christopherson collected at the post-mortem examination of a native of Khartoum an *Acanthocephala* which he was good enough to forward to us in London. The rostellum was absent, but there can be little doubt that the specimen belongs to this species, with which it conforms in size and external marking.

RECENT STATISTICS OF THE OCCURRENCE OF INTESTINAL PARASITES IN MAN.

THE following table is compiled from a number of recent publications, giving the results of careful microscopical examination of the fæces for the eggs of helminths :—

				China.		
Locality	Martinique * (F. Noc).	Barbary * (H. Foley).	Ivory Coast * (F. Sorel).	Hankow † (Drs. Hodge & Booth).	Shanghai † (Dr. Day).	Anking † (Dr. Taylor).
Number of individuals examined.	225	214	320	147	100	67
	per cent.	per cent.	per cent.	per cent.	per cent.	per cent.
Ascaris lumbricoides . .	44	83	1·7	70	54	70
Trichocephalus trichiuris . .	38	13	6·8	40	21	28
Ankylostomes	36·8	none	75	11	15	36
Schistosomum japonicum	—	—	—	8	—	16
Schistosomum hæmatobium . .	16	—	—	—	—	—
Tænia saginata	—	2·8	—	—	—	—
Tænia solium	several	—	—	—	—	—
Tænia sp. inq.	—	—	2·4	—	—	—
Hymenolepis nana	—	4·2	—	—	—	—
Hymenolepis diminuta	0·4	—	—	—	—	—
Opisthorchis sinensis	—	—	—	—	1	—
Oxyurias vermicularis	several	rare	—	7	—	—

* Bull. de la Soc. Path. Exotique, 1911.

† China Medical Journal, 1910.

STALE-MATE IN THE BILHARZIA CONTROVERSY: AND A NEW THEORY.

[A. Looss.—Journal of Tropical Medicine and Hygiene, June 15th, 1911.]

IN a highly interesting communication Prof. Looss brings to a close, it is to be hoped, the recent controversy upon the significance of the "lateral spine" sometimes met with in eggs from cases of African Bilharziasis. He conclusively demonstrates that Dr. Sambon's "species" and his own "hypothesis" are untenable. Fortunately he is able at the same time to offer a new and much simpler explanation, which will in all probability be heartily welcomed, as it dispenses with the somewhat far-reaching postulates which burdened his earlier view, viz.:—(*a*) the egg develops parthenogenetically; (*b*) the miracidium penetrates the skin; (*c*) the cercaria from one sporocyst are all of the same sex.

In 1909 Goldschmidt showed that the so-called yolk-cells are really shell-substance-carrying cells. Adopting this view Looss proceeds to suppose that for the formation of normal, *i. e.* terminal spined, eggs just sufficient yolk-cells are admitted to the ootype to fill the cavity proper. When the shell forms it assumes the shape of the cavity of the ootype, the subsequent protrusion of a small portion of the shell-substance along the funnel giving rise to the terminal spine, which remains soft and mouldable for a time. When the eggs are formed in quick succession, as in normal adults, they pass on into the uterus, assuming there their definite shape. If, however, the yolk-cells accumulate, for some reason or other, in the ootype to such an extent that they distend its cavity, bulging particularly the dorsal wall, they fill not only the funnel-shaped portion, but frequently also a part of the short duct connecting the ootype with the uterus; and if the egg were to remain in the ootype until the shell had completely hardened, then any irregularity in shape would be retained, and in this way there would result the various forms known as "lateral-spined" or, as Prof. Looss suggests, "ventral-spined" eggs. In the same article Prof. Looss states that he has been able to find the "lateral spine" described by Leiper in *Schistosoma japonicum* eggs in only 75 per cent. of those examined. R. T. L.

ON THE PASSAGE OF ANKYLOSTOME LARVÆ FROM LUNGS TO INTESTINE.

[FÜLLEBORN and SCHILLING-TORGAU.—Archiv für Schiffs u. Tropen-Hygiene, vol. xv. p. 569, Sept. 1911.]

PROFESSOR FÜLLEBORN, with the surgical assistance of von Schilling-Torgau, has carried out a series of ingenious experiments upon dogs to determine definitely whether or not the contention of Sambon that in Ankylostomiasis the larvæ reach the intestinal wall by the blood-stream, and not, as Looss is held to have demonstrated, by their passage from the lungs up the trachea and down the œsophagus to the intestines. Tracheotomy was performed on a number of dogs. The lower part of the trachea had a canula inserted and the passage of larvæ from the lungs to the œsophagus rendered impossible. Some of the dogs were then heavily infected with larvæ of *Ancylostomum caninum* and others with *Strongyloides stercoralis*. After some little time the secretion collected from the canula in the trachea swarmed with larvæ. In other dogs the œsophagus, instead of the trachea, was cut across and the ends implanted in the skin: in these the larvæ escaped from the upper cut end. As a control, dogs which had not been operated upon were similarly infected, and in these the resulting intestinal infection was intense. In the dogs which had been operated upon, however, the bulk of the larvæ escaped from the cut ends of the trachea or œsophagus; nevertheless the animals did not remain entirely free from infection. A small number of parasites were found later in the gut, indicating that a certain number of the larvæ had been able to pass through the lungs into the systemic circulation and so reach the gut wall. The experiments demonstrate that for all practical purposes Looss' original contention holds good. The larvæ that escape from the lungs and pass up the trachea are not "side-tracked" harmless forms as Sambon maintained, but are actually on their way to their final destination.

Moreover, Fülleborn shows that these migrating forms are undergoing rapid metamorphosis, so that they are probably better able to withstand the changes in media through which they pass. As the experimental animals did not remain entirely free from infection, Sambon's alternative route must be recognized as a possible albeit unimportant one.

Professor Fülleborn records in his brief preliminary note two further observations of interest. He finds that even when filariform Strongyloides larvæ are taken direct through the mouth, they penetrate the stomach wall and make the circuit of lungs, trachea, and œsophagus just in the same way as those entering through the skin. Secondly, the filariform larvæ are occasionally able to attain maturity—as parthenogenetic females—in the trachea, and give rise there to rhabditic embryos, thus approaching the condition normally found in *Rhabdonema nigrovenosum* (*Rhabdias bufonis*), the Nematode so commonly met with in the frog's lung. R. T. L.

HELMINTHIC INFECTIONS IN PANAMA.

[DARLING.—Bulletin de la Société Pathologique Exotique, vol. iv, p. 5, May 1911.]

THE incidence of intestinal worms in native and West Indian male and female insane patients at Ancon Hospital, Canal Zone, is shown by the use of special methods to be as follows:—

No. of Patients examined	285	
Negative examinations of stools . .	29·6	per cent.
Hookworm infections	37·9	,,
Whipworm infections	46·7	,,
Roundworm infections	4	,,
Strongyloides infections	20	,,
Oxyuris infections	1·8	,,

Darling adds that all the Cestode infections—viz., *Tænia saginata*, *T. solium*, *T. echinococcus*, and *Hymenolepis nana*—are confined to Europeans and Americans, and *Schistosomum hæmatobium* to natives from the West Indies. R. T. L.

PARASITES OF DOGS IN THE PUNJAUB.

[Journal of Tropical Veterinary Science, vol. vi. no. 3, 1911.]

MR. S. H. GAIGER, of the Punjaub Veterinary College, gives the following useful table of the relative frequency of the parasites found in 50 pariah dogs in Lahore:—

Linguatula tœnioides	19	38 per cent.
Spiroptera sanguinolenta	18	36 ,,
Ascaris marginata	19	31 ,,
Tœniæ spp.	45	90 ,,
Ankylostoma	19	38 ,,
Opisthorchis noverca	19	38 ,,

The common occurrence of *O. noverca* is of especial interest, as it has on two occasions been found in man.

Helpful descriptions and illustrations of *Thelazia callipæda*, Railliet and Henry, 1910, recently found in the dog, also appear in the same article.

R. T. L.

CHECK-LIST OF HELMINTHES PARASITIC IN EQUINES †.

By ROBERT T. LEIPER, D.Sc., M.B., F.Z.S.,
HELMINTHOLOGIST TO THE LONDON SCHOOL OF TROPICAL MEDICINE.

Species marked thus * are represented in the Collection of the Helminthological Department of the School.

Order **NEMATODA.**

Non-Bursata.

Family I. ASCARIDÆ.

Genus ASCARIS, Linnæus, 1758.

**Ascaris megalocephala*, Cloquet, 1824.

Family II. OXYURIDÆ.

Genus OXYURIS, Rudolphi, 1803.

(a) **Oxyuris curvula*, Rudolphi, 1803.
(b) **Oxyuris mastigodes*, Nitzsch, 1866.
(c) *Oxyuris poculum*, v. Linstow, 1904.
(d) *Oxyuris tenuicauda*, v. Linstow, 1900.

Genus PROBSTMAYRIA, Ransom, 1907.

**Probstmayria vivipara* (Probstmayer), 1865.

Family (inq.).

Genus PTEROCEPHALUS, v. Linstow, 1899.

**Pterocephalus viviparus*, v. Linstow, 1899.

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† Read at the National Veterinary Association, Carnarvon, July 26th, 1911.

Family III. FILARIIDÆ, Braun, 1895.

Subfamily Filariinæ, Stiles, 1907.

Genus Filaria, Müller, 1787.

(a) *Filaria cordicola*, v. Linstow, 1905.
(b) **Filaria equina*, Abildg., 1789.
(c) *Filaria multipapillosa*, Condamine & Drouilly, 1878.
(d) *Filaria inermis*, Grassi, 1887.

Genus Dermofilaria, Rivolta, 1884.

Dermofilaria irritans, Rivolta, 1884.

Subfamily Dracunculiinæ, Stiles, 1907.

Genus Dracunculus, Kniphoff, 1759.

**Dracunculus medinensis* (Linnæus), 1758.

Subfamily Onchocercinæ, Leiper, nom. nov.

Genus Onchocerca, Diesing, 1841.

(a) **Onchocerca reticulata*, Diesing, 1841.
(b) *Onchocerca cervicalis*, Railliet & Henry, 1910.

Family IV. SPIRURIDÆ.

Genus Spiroptera, Rudolphi, 1819.

(a) **Spiroptera megastoma*, Rudolphi, 1819.
(b) **Spiroptera microstoma*, Rudolphi, 1819.

Genus Thelazia, Bosc, 1819.

**Thelazia lachrymalis* (Gurlt), 1831.

Genus Gongylonema, Molin, 1857.

Gongylonema scutata (Müller), 1869.

Bursata Vera.

Family V. STRONGYLIDÆ.

Subfamily Strongylinæ.

Genus Strongylus.

(a) **Strongylus equinus*, Müller, 1784.
(b) **Strongylus edentatus* (Looss), 1900.
(c) **Strongylus vulgaris* (Looss), 1900.

Genus Triodontophorus, Looss, 1901.

(a) **Triodontophorus minor* (Looss), 1900.
(b) *Triodontophorus intermedius*, Sweet, 1908.
(c) **Triodontophorus serratus* (Looss), 1900.

Genus Gyalocephalus, Looss, 1900.

**Gyalocephalus capitatus*, Looss, 1900.

Genus CYLINDROPHARYNX, Leiper, 1911.

(a) **Cylindropharynx brevicauda*, Leiper, 1911.
(b) **Cylindropharynx longicauda*, Leiper, 1911.

Genus CYLICOSTOMUM, Railliet, 1900.

(a) *Cylicostomum alveatum*, Looss, 1900.
(b) **Cylicostomum auriculatum*, Looss, 1900.
(c) **Cylicostomum bicoronatum*, Looss, 1900.
(d) **Cylicostomum calicatum*, Looss, 1900.
(e) *Cylicostomum catinatum*, Looss, 1900.
(f) *Cylicostomum coronatum*, Looss, 1900.
(g) **Cylicostomum elongatum*, Looss, 1900.
(h) **Cylicostomum labiatum*, Looss, 1901.
(i) **Cylicostomum labratum*, Looss, 1900.
(j) **Cylicostomum nassatum*, Looss, 1900.
(k) **Cylicostomum poculatum*, Looss, 1900.
(l) **Cylicostomum radiatum*, Looss, 1900.
(m) **Cylicostomum tetracanthum* (Mehlis), 1831.

Genus ŒSOPHAGODONTUS, Railliet & Henry, 1902.

Œsophagodontus robustus (Giles), 1892.

Family VI. METASTRONGYLIDÆ.

Subfamily TRICHOSTRONGYLINÆ, Leiper, 1908.

Genus TRICHOSTRONGYLUS, Looss, 1905.

Trichostrongylus axei (Cobbold), 1886.

Subfamily METASTRONGYLINÆ, Leiper, 1908.

Genus DICTYOCAULUS, Railliet & Henry, 1900.

**Dictyocaulus arnfieldi* (Cobbold), 1884.

Pseudo-Bursata.

Family VII. EUSTRONGYLIDÆ.

Genus DIOCTOPHYME, Collet-Megret.

Dioctophyme renale (Goeze), 1782.

Order **TREMATODA.**

Family VIII. FASCIOLIDÆ, Railliet, 1895.

Subfamily FASCIOLINÆ, Looss, 1899.

Genus FASCIOLA, Linnæus, 1758.

**Fasciola hepatica*, Linnæus, 1758.

Family IX. PARAMPHISTOMIDÆ, Fischoeder, 1900.

Subfamily CLADORCHINÆ, Fischoeder, 1900.

Genus PSEUDODISCUS, Sonsino, 1895.

**Pseudodiscus collinsi* (Cobbold), 1875.

Genus GASTRODISCUS, Cobbold, 1877.

(a) **Gastrodiscus ægyptiacus* (Cobbold), 1876.
(b) *Gastrodiscus secundus*, Looss, 1907.

Family X. SCHISTOSOMIDÆ, Looss, 1899.

Genus SCHISTOSOMUM, Weinland, 1858.

**Schistosomum indicum*, Montgomery, 1906.

Order **CESTODA**.

Family XI. TÆNIIDÆ.

Genus TÆNIA, Linnæus, 1758.

(a) **Tænia cœnurus*, larval stage (*Cœnurus cerebralis*).
(b) *Cysticercus fistularis* (? *Cysticercus tenuicollis*).

Genus ECHINOCOCCIFER, Rudolphi, 1801.

**Echinococcifer echinococcus* (Siebold), 1853, larval stage (Hydatid).

Family XII. ANOPLOCEPHALIDÆ.

Genus ANOPLOCEPHALA, E. Blanchard, 1848.

(a) **Anoplocephala mamillana* (Mehlis), 1831.
(b) **Anoplocephala perfoliata* (Goeze), 1782.
(c) **Anoplocephala plicata* (Zeder), 1800.

	Families.	Subfamilies.	Genera.	Species.
Nematoda	7	6	10	45
Trematoda	3	2	4	5
Cestoda	2	–	3	6
Helminthes	12	8	17	56

ALPHABETICAL LIST OF SPECIES PARASITIC IN EQUINES.

ægyptiacus (*Gastrodiscus*).
alveatum (*Cylicostomum*). [*Cylichnostomum*, *Cyathostoma*.]
**arnfieldi* (*Dictyocaulus*). [*Strongylus*.]
**auriculatum* (*Cylicostomum*). [*Cylichnostomum*, *Cyathostoma*.]
axei (*Trichostrongylus*). [*Strongylus*.]
**bicoronatum* (*Cylicostomum*). [*Cylichnostomum*, *Cyathostoma*.]
**brevicauda* (*Cylindropharynx*).
**calicatum* (*Cylicostomum*). [*Cylichnostomum*, *Cyathostoma*.]
**capitatus* (*Gyalocephalus*).
catinatum (*Cylicostomum*). [*Cylichnostomum*, *Cyathostoma*.]

*_cerebralis_ (_Cœnurus_).
cervicalis (_Onchocerca_).
*_cœnurus_ (_Tænia_).
*_collinsi_ (_Pseudodiscus_). [_Amphistomum._]
cordicola (_Filaria_).
coronatum (_Cylicostomum_). [_Cylichnostomum, Cyathostoma._]
*_curvula_ (_Oxyuris_).
*_echinococcus_ (_Echinococcifer_). [_Tænia._]
*_edentatus_ (_Strongylus_). [_Sclerostomum._]
*_elongatum_ (_Cylicostomum_). [_Cylichnostomum, Cyathostoma._]
*_equina_ (_Filaria_).
*_equinus_ (_Strongylus_). [_Sclerostomum._]
fistularis (_Cysticercus_).
gigas (_Eustrongylus_). See _renale._
*_hepatica_ (_Fasciola_). [_Distomum._]
*_indicum_ (_Schistosomum_).
inermis (_Filaria_).
intermedius (_Triodontophorus_).
irritans (_Dermofilaria_). [_Filaria._]
*_labiatum_ (_Cylicostomum_). [_Cylichnostomum, Cyathostoma._]
*_labratum_ (_Cylicostomum_). [_Cylichnostomum, Cyathostoma._]
*_lacrymalis_ (_Thelazia_). [_Filaria._]
*_longicauda_ (_Cylindropharynx_).
mamillana (_Anoplocephala_). [_Tænia._]
*_mastigodes_ (_Oxyuris_).
*_medinensis_ (_Dracunculus_). [_Filaria._]
*_megalocephala_ (_Ascaris_).
*_megastoma_ (_Spiroptera_). [_Filaria._]
*_microstoma_ (_Spiroptera_). [_Filaria._]
*_minor_ (_Triodontophorus_).
multipapillosa (_Filaria_).
*_nassatum_ (_Cylicostomum_). [_Cylichnostomum, Cyathostoma._]
*_perfoliata_ (_Anoplocephala_). [_Tænia._]
*_plicata_ (_Anoplocephala_). [_Tænia._]
*_poculatum_ (_Cylicostomum_). [_Cylichnostomum, Cyathostoma._]
poculum (_Oxyuris_).
*_radiatum_ (_Cylicostomum_). [_Cylichnostomum, Cyathostoma._]
renale (_Dictophyme_). [_Eustrongylus._]
*_reticulata_ (_Onchocerca_). [_Filaria, Spiroptera._]
robustus (_Œsophagodontus_). [_Sclerostomum._]
scutata (_Gongylonema_).
secundus (_Gastrodiscus_).
*_serratus_ (_Triodontophorus_).
*_sonsinoi._ See _ægyptiacus_ [_Gastrodiscus_].
tenuicauda (_Oxyuris_).
*_tetracanthum_ (_Cylicostomum_). [_Strongylus, Sclerostomum._]
*_vivipara_ (_Probstmayria_). [_Oxyuris._]
*_viviparus_ (_Pterocephalus_).
*_vulgaris_ (_Strongylus_). [_Sclerostomum._]

A NEW ECHINOSTOME PARASITE IN MAN.

By R. T. LEIPER, D.Sc., M.B.,

HELMINTHOLOGIST TO THE LONDON SCHOOL OF TROPICAL MEDICINE.

DURING the past summer (1911) I have received for report two consignments of flukes collected from the intestine of tamils in the Malay States. The first set was forwarded by Dr. Macaulay, of Singapore, the second by Dr. Stanton, of the Kuala Lumpur Institute for Medical Research. At first glance I took them to be immature forms of *Fasciolopsis buski,* the colour, general shape, and wide ventral sucker corresponding quite closely.

When the specimens were cleared in creosote, however, it was noticed that the skin was thickly beset with stout acicular spines, and at the anterior the spines had a disposition that is quite characteristic of the family *Echinostomidæ*. In the uterus of some specimens large brown eggs were distinguished. The only member of the *Echinostomidæ* found hitherto in man is Garrison's *Fascioletta ilocana,* which, as Odhner has recently demonstrated, is a member, not of the family *Fasciolidæ* as Garrison supposed, but of *Echinostomidæ* and of the genus *Echinostomum.*

The specimens received were all more or less coiled up, the dorsal being the outer surface, and on this account were somewhat difficult to examine satisfactorily *in toto.* The measurements of body given here are based upon the most easily handled example. The description of the internal structure, as seen in section, is based upon a series of paraffin sections made from one of the coiled forms.

The body is thick and fleshy, measuring 12 mm. in length, 3 mm. in greatest breadth, and 1·3 mm. in thickness. The specimens, preserved in spirit, are of a light brown colour. The general outline of the body recalls that of *Opisthorchis felineus.* Both ends of the body are bluntly rounded. At the anterior end a furrow on either side, extending inwards for about a third of the total body breadth at this point, detaches to some extent a kidney-shaped portion carrying the oral sucker. Along the edge of this circum-oral disc is a distinct row of stout spines which continue unbroken round the dorsum of the worm, although the furrow is limited to the ventral side. These spines number 42 in all, of which three on either side form a distinct set. These latter are situated at the inner angle of each lateral lapel of the circum-oral disc, for the furrow and row of spines do not continue across the middle line on the ventral surface as on the dorsum. The spines vary in size, those on the ventral side are 0·07 mm. in length, whilst on the dorsal the smaller are 0·05 × 0·016 mm. The cuticle covering the body behind the pathognomonic hoop of spines is also furnished with spines, but these are not distributed uniformly over the whole surface, for whereas on the ventral surface they extend backwards to the posterior end of the body, on the dorsal aspect they are limited to a triangular area which terminates at the level of the upper edge of the ventral sucker.

Alimentary canal.—The oral sucker occupies a third, the middle third, of the total diameter of the circum-oral disc. In sagittal section the greatest thickness of the muscular wall is 0·07 mm. The oral sucker communicates with the spheroid muscular pharynx by a thin-walled portion, 0·175 mm.

long. The pharynx is spheroidal and has a diameter of 0·25 mm., and the wall is 0·12 mm. deep in its thickest part.

The œsophagus measures 0·04 mm. in length and bifurcates immediately behind the pharynx.

The main gut branches are simple and run to end blindly at the posterior end of the body.

The ventral sucker, which is several times larger than the oral sucker and measures 0·9 × 0·75 mm., has a large orifice, oval in shape, with the greater diameter transverse. Its cavity, 0·7 mm. deep, is saccular and lies with its longitudinal diameter at an angle of about 40° with the ventral surface. The muscular wall in section is from 0·2 to 0·25 mm. thick.

Genitalia.—The testes are tandem and lie in the middle of the body behind the ventral sucker. They are deeply lobed.

The cirrus pouch is well developed and reaches behind to the level of the fundus of the ventral sucker. A long thick cirrus extrudes from the genital pore which lies in the angle between the neck and the anterior lip of the ventral sucker.

The ovary has a smooth surface, measuring in section 0·3 mm. in antero-posterior diameter, and lies immediately in front of the anterior testis, 0·85 mm. behind the ventral sucker.

The yolk-glands are very numerous and stretch from the level of posterior limit of the ventral sucker to the posterior end of the body, where the acini of either side become indistinguishable from one another.

The eggs are few in number, brown in colour, and of large size.

It does not seem probable that this Echinostomum is a normal parasite of man, and its presence in domesticated animals, particularly in cats and dogs in the Malay States, should be sought. On the other hand, the form above described does not resemble the Echinostoma that have been recorded from these animals, nor indeed, so far as I can determine, to any known species. The number of Echinostomes known in mammals is very small. From *E. trigonocephalum*, the types of which I have recently examined, the new form is distinguished by general size, extension of yolk-glands, and number of circum-oral spines. On similar grounds I also differentiate *E. perfoliatum*, Ratz, 1908 (*E. gregale*, Railliet), and *E. spiculator* (Duj.). For the new species I propose the name *Echinostoma malayanum*.

A METHOD FOR DEALING WITH TOWN WELLS INFECTED WITH GUINEA-WORM.

By R. T. LEIPER, D.Sc., M.B., F.Z.S.

It has been demonstrated, in India, that wells contaminated with guinea-worm remain infective for many months. As over 90 per cent. of the guinea-worm eruptions occur on the lower extremities, particularly in the region of the ankle, it is easy, theoretically at least, to prevent infection of uncontaminated wells by the erection of a fairly high parapet around the mouth of the well, and by cutting off the possibility of return of drawn water. It has appeared a more difficult problem to devise a safe and efficient method of dealing with a well already contaminated or under suspicion. Its permanent closure may involve much hardship, especially in times of drought. The addition of chemicals may render the water unpalatable or unpotable.

To render the water innocuous the infective stage of the guinea-worm, *i. e.*, the metamorphosed larva within the *cyclops*, must be destroyed. I have shown some years ago that when the infected *cyclops* dies the encysted worm does not become free but dies also. If, then, one could destroy the adult *cyclops* in a well suspected of guinea-worm contamination, its water would be rendered innocuous, and if surrounded with a parapet, could be kept so, even although a new brood of *cyclops* appeared.

Some laboratory experiments have suggested to me the idea that it would be comparatively easy to kill off the adult *cyclops* in a well without in any way interfering with its continued use. I find that when the temperature of the water in which *cyclops* are living is suddenly raised several degrees they die almost immediately. In England it is only necessary to raise the temperature from 15° C. to 35° C. in order to ensure a fatal result.

The water in a well could be heated to the necessary degree by passing steam through it from a portable boiler. This method of heating is in general use in dairies in the making of cheese. An apparatus similar in all respects to that used at the present time for the cleansing of stone buildings by a steam-jet might be adopted. The nozzle of the steam-pipe should reach deep into the water of the well to obtain the best result. My friend Mr. Stanley Phillips, B.Sc., Lecturer on Physics in the Birmingham Municipal Technical School, has kindly given me the following formulæ :—

To raise 1000 gallons of water from t° C. to T° C. (say 15° C. to 65° C.) :

t = temperature of water in the well.

T = temperature to which it is to be raised.

Now, 1 gallon of water = 10 lbs. = 10 × 450 grammes,

so that heat required for y gallons will be

$$y \times 10 \times 450 \times (T - t) \text{ calories} \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \qquad (1)$$

This heat is to be supplied by condensation of x grammes of steam.

Now, 1 gramme of steam at 100° C. in being condensed and then reduced to temperature T° gives out 540 + (100 − T) calories.

Therefore x grammes give out

$$x[540 + (100 - T)] \text{ calories} \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \qquad (2)$$

Equating (1) and (2) we have

$$x[540 + (100 - T)] = 10 \times 450 (T - t) \times y.$$

or
$$x = \frac{y \times 10 \times 450 (T - t)}{540 + (100 - T)} \text{ grammes,}$$

i. e.,
$$x = \frac{y (T - t)}{540 (100 - T)} \text{ gallons.}$$

(If t = 15° C., T = 65° C., and y = 1000 gallons, then

$$x = \frac{1000 \times 50}{540 + 35} = 87 \text{ gallons.})$$

Thus to purify a well of 1000 gallons by raising its temperature from 15° C. to 65° C. would take 87 gallons of water as steam.

As regards the size of the boiler we can approximate by considering the case of one heated by coal.

Each gallon of water which is boiled up requires about 1 lb. of coal, so that for the above well. under conditions stated. there will be required 87 lbs. of coal (or its equivalent in oil).

For every square foot of grate the coal can be burned at from 10 to 20 lbs. per hour (say 15 lbs. per hour), so that if the grate is 1 sq. foot it would take $\frac{87}{15}$ hours. *i. e.* almost six hours.

To evaporate at the rate of 90 gallons in half an hour would require a grate twelve times as large. *i. e.* 12 sq. ft.

If the above method, based merely on laboratory experiments. should prove practicable in tropical countries on the larger scale suggested. it would only need to be applied once to each well if precautions were taken afterwards against fresh contamination. But even if it were considered desirable to visit the wells periodically. we know, from the period of development the parasite requires to undergo before it reaches the infective stage, that these visits need not be more frequent than monthly. Moreover, it is well known that there is a marked seasonal incidence on the occurrence of *Dracontiasis.* During the dry season the natives probably resort more frequently to wells than at other times. and during these periods also the water in the wells becomes low and the sick infected *cyclops.* which do not swim about so actively and keep nearer the bottom. are now taken up in the draw-bucket. At the time when the surface water in the small ponds has disappeared (save where it is thickly overgrown with vegetation. and such collections show that they have been overlooked by the authorities). and the calls upon the sanitary service have somewhat relaxed, attention can be directed profitably to the visitation of the town wells.

IV.—PATHOLOGY.

By H. B. NEWHAM.

The Question of Flies as Leprosy Carriers.

By E. P. MINETT, M.D., D.P.H., D.T.M. & H.,
Assistant Government Bacteriologist, British Guiana.

Now that the infectivity of Leprosy, as against its inheritance, is accepted, researches on the subject are rightly directed principally to finding the agent or agents by which the infection is brought about.

The cause of the disease has been proved to be the *Bacillus lepræ* of Hansen, but the difficulty of procuring its growth on artificial media has so far proved insuperable. I have personally tried all the published methods, old and new, for its culture in the laboratory, with and without various modifications of my own, but have been as yet quite unsuccessful in obtaining a growth. Deycke's Streptothrix seems so far to be the nearest approach to an artificial growth. It has been impossible to produce leprosy in any animal by means of an artificial culture of the leprosy bacillus, and for obvious reasons direct inoculation from man to man is not possible experimentally.

For the last ten years experiments have been directed chiefly towards finding that agent concerned in the spread of so many infectious disorders, a carrier, parasitic or otherwise, and much work has been done on the subject.

So far back as 1650 Admiral Henrik Bjelke*, Governor of Iceland, reports that "Leprosy is increasing rapidly in Iceland, because of the filthy dwellings and want of personal cleanliness of the people." This would seem to lend support to the idea that leprosy might be transmitted by vermin, and may be taken as one of the earliest suggestions in this direction.

In 1900 the possibility of transmission by blood-sucking insects was first considered. Palm † expressed the opinion that it was transmitted by the ordinary body-louse (*Pediculus vestimentorum*), but he adduced no evidence either clinical or experimental. Scott ‡, from his experience in the Sandwich Islands and Madeira, believed that mosquitoes acted as carriers. Noc § found some bacilli in mosquitoes, and attributes the long and variable latent period to the presence of a resistant or spore form of the bacillus, possibly in the mosquito; but, so far as I am aware, no spore form of leprosy has ever been demonstrated, nor has the resistance of the bacillus to adverse conditions of life been proved to be great compared with the spore stage of anthrax or the vegetative stage of the Gaertner group.

Cazamian ‖, in China, also attributed its spread to mosquitoes.

In 1901 Joly ¶, in Madagascar, thought that mosquitoes were not responsible, or Europeans would suffer more than natives, instead of less, but that flies were more likely to transmit the disease by infecting wounds and mucous surfaces. He observes further that the European who is clothed and attentive to small wounds, and who drives off flies as much as possible, runs, on this account, much less risk of infection than does the native who pays far less attention to these points. Fish, he thinks, may possibly be infected by flies during the process of drying; and Hutchinson's fish theory is of course well known, although the fact that leprosy is common in some parts of the Soudan where fish is unknown has been evidenced against it. On the other hand, flies and other blood-sucking parasites are abundant in that region.

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* Lovsamling for Island.

† 'Lancet,' Aug. 18, 1900.

‡ 'Lancet,' Sept. 29, 1900.

§ Ann. d'Hyg. et de Méd. colon., juillet-sept. 1903, p. 481, et janvier-mars, 1904, p. 11.

‖ Arch. de méd. navale, déc. 1904, p. 452.

¶ *Ibid.* juin 1901, p. 459.

Leprosy certainly increases in time of famine, but so do dirt, skin-disease, and suppurating wounds—all predisposing to an infection with the bacillus lepræ; and its spread in small homes rather than in hospitals may be due to the presence of scabies and pediculi in the former.

Choksy * attributes its spread to the bites of mosquitoes and fleas; while Kermorgant †, in 1902, states that cats get a form of anæsthetic leprosy, and suggests the rat-flea (*Pulex cheopis*) as the possible carrier.

Thiroux ‡, 1903, considers that jigger (*Sarcopsilla* or *Pulex penetrans*) wounds may be a site of infection, and instances swelling of the feet as an early indication of leprosy. It has also been recorded in the Dutch West Indies that in 5 per cent. of the leprosy cases the first symptoms appear in the feet of persons accustomed to go bare-footed. As, however, swelling of the feet and ankles is frequently present in cases of jigger, it would seem far more probable that the swelling is the result of a secondary septic infection of the original puncture, especially as various organisms, such as *Staphylococcus aureus*, *Streptococcus*, and *Bacillus coli communis*, are frequently present in these wounds. I have made many bacteriological examinations of leprotic ulcers, and have always succeeded in isolating, in addition to the leprosy bacillus, many organisms, principally *Staphylococcus aureus* and *albus*, but occasionally *Streptococcus pyogenes longus* and *B. coli communis*, and yet the presence of these organisms does not appear to influence the presence of the leprosy bacillus in any way.

In 13 cases examined, leprosy bacilli were present in the serum and exudate from these ulcers in enormous numbers, together with the pathogenic organisms above mentioned. My own opinion is that it is the presence of these organisms, setting up a secondary infection, which causes the ulcers to exacerbate so freely, because when the ulcers are cleaned and covered with an antiseptic dressing, in a few days the inflammation subsides and the pathogenic organisms disappear, although the leprosy bacilli are still present in apparently undiminished numbers. The ulcers themselves can then be healed by painting with a weak solution of nastin in aseptic oil, preferably one of mineral composition.

Von Bassewitz § and Mugliston ‖ accuse the *Acarus scabei* of being a possible carrier of leprosy, and cite in support of their theory the fact that itch is frequent amongst lepers.

Goodhue ¶, in 1905, claims to have found the leprosy bacillus in the female culex and in *Cimex lectuarius*.

Römer ** found bacilli in various arachnoides, but not in the culicides or pediculi. It is said that Indian gurus have been known to take bugs from the persons of lepers and sell them with the object of transmitting leprosy by their means.

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* Report on Leprosy and the Homeless Lepers' Asylum, Matunga, Bombay, 1901.

† 'Lepra,' iii. p. 13.

‡ Ann. d'Hyg. et de Méd. colon., 1903, p. 564.

§ Münch. med. Wochschr. 1905, no. 41.

‖ Journal of Tropical Medicine and Hygiene, 1905, July 15. ¶ *Ibid.* 1906, Sept. 15.

** "La Lepre," Bull. de l'Acad. Roy. de Med. de Belgique, 1906, Jan. 27, p. 99.

Ehlers *, of Copenhagen, experimented with cimex, especially after casting their skins, and detected the bacilli after the insect had been allowed to suck the blood of lepers. Marchaux and Bourret † also experimented in a similar manner with fleas.

With regard to the above-mentioned experiments, it is known that filaria can be detected in the stomach contents of bugs fed on filarious patients, so that the evidence in favour of blood-sucking insects as possible carriers of leprosy is rapidly accumulating, but for obvious reasons it is impossible to attempt to prove this theory of transmission from man to man by direct experiment.

With a view to showing, if possible, that the leprosy bacillus is carried about by the common house-fly (*Musca domestica* and *lucilia*), as in a similar manner it has been proved that various other diseases, such as enteric and cholera, are carried, a number of flies were captured at the Leper Asylum, Mahaica, British Guiana, on two occasions. These flies were kept in a sterile bottle for several days and were then allowed to feed on the ulcerated patches present on several lepers for a short time before returning to the sterile bottle. They were then kept for a period of three days before examination. The method I adopted was as follows:—The flies were first killed with chloroform vapour: each fly was then placed in a thin sterile test-tube containing 4 c.cm. of sterile saline solution. They were allowed to soak in this for 48 hours, with frequent shakings, care being taken not to detach legs or wings. At the end of this period the fly was removed and the saline placed for five minutes in a high-speed electric centrifuge. The supernatant fluid was carefully drawn off with a pipette, and the deposit placed on a sterile glass slide and allowed to evaporate in the air, covered by a watchglass. This was then stained with Ziehl-Neelsen's differential stain and examined for the presence of bacilli. In this manner, of thirty flies examined, in one instance leprosy bacilli were detected.

The same flies were then placed in fresh sterile test-tubes, and their intestines teased out by means of sterile platinum needles, care being taken to break up the intestine itself and to express the faeces. Four c.cm. of sterile saline solution was then added to each tube, which was allowed to stand for 24 hours with frequent shakings. At the end of this period the fragments of fly were removed by means of platinum loops, the fluid centrifugalised, and the deposit dried, stained, and examined as before. Of thirty specimens examined in this manner, four specimens contained well-formed leprosy bacilli, and one specimen showed their presence in a very degenerated form.

From these experiments it would seem that flies must certainly be reckoned with as possible factors in the dissemination of leprosy, if only as mechanical carriers: for it would be extremely easy for a fly, having previously fed on a leprous ulcer, to convey infection to others, either by way of cuts and abrasions on the skin, or possibly by the mucous membrane of the nasal cavity.

Also the fact that undegenerated leprosy bacilli showing no plasmolysis, or other evidences of destruction, were detected in the fly's intestine after a period of three days, would seem to point to the conclusion that the waxy coating present on these bacilli is extremely resistant to the gastric juices, and that the organisms are presumably capable of conveying the infection to a healthy person if planted on a suitable medium, such as the serum present on a cut or ulcer.

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* 'Lepra Konferenz,' vol. iii. p. 29.

† Ann. de l'Institut Pasteur, vol. xix. p. 389.

So far as I am aware, the non-biting Muscidæ have not yet been suggested as possible carriers of leprosy. That they may spread infection has been amply demonstrated by Orton and Dodd * who, working with *Bacillus prodigiosus,* planted cultures in the laundry o a hospital, and later detected the same bacillus in the scullery and five screened ward dining-rooms, at intervals of from two to six days after the original plant. From these experiments they conclude it is impossible to keep the flies from access to fæcal matter, food, &c.

With a view to testing the possibility of leprosy infection by means of flies' fæces, I obtained 30 flies of the ordinary non-biting Muscidæ species. These were placed in sterile glass roll culture-tubes, plugged with cotton-wool. They were kept 48 hours without food, and then fed on the serum, known to be rich in bacilli, exuded from a freshly-cut leprotic nodule. This was effected by making a fairly deep cut in a suitable nodule, scraping the sides, and then placing the mouth of the tube containing the flies over the same. It was found that the flies fed on the serum and blood readily. They were allowed to feed in this manner twice daily for seven consecutive days. At the end of this period the flies were killed with chloroform vapour and shaken out of the tubes. Ten c.cm. of sterile 0·75 per cent. saline solution was placed in each tube, and the fæces thoroughly shaken up and allowed to remain for 24 hours to soak; then shaken up again, the saline centrifugalised in sterile tubes, and the deposit collected on slides, dried, and stained as before. In order to concentrate the deposit, the saline obtained from 5 flies was mixed, making a total of 6 slides.

On examination the results were as follows:—

2 slides showed many typical bacilli present;
2 slides showed many typical bacilli, also a large number of bacilli showing well-marked granular destruction;
1 slide showed well-marked granules but no typical bacilli;
1 slide was negative.

With the object of confirming my former experiments, I placed 10 flies in each of four sterile glass jars covered with mosquito netting. The flies were then fed daily for two weeks on serum from a leprotic nodule. At the end of this period they were removed and examined both externally and internally for the presence of leprosy bacilli. These were found to be present. The sterile bottles were then carefully washed out with saline fluid and the fæces only examined for bacilli. In all four cases large numbers of leprosy bacilli were detected, showing no evidence whatever of degeneration.

As the bottles were examined three days after removal of the flies themselves, I regard this as most important, showing as it does that leprosy bacilli can be detected in a practically unchanged state in flies' fæces after a period of several days, and most probably longer.

These experiments agree with those carried out by Currie †; but as they are carried a stage further, and demonstrate the presence of the bacilli in fæces at least three days old, I have ventured to record them as confirmatory evidence.

* Journal of Tropical Medicine and Hygiene, Jan. 16, 1911.

† United States Public Health Bulletin, no. 39, Sept. 1910.

Unfortunately, I am not able to definitely prove the lepra bacilli present in the flies' fæces to be capable of causing infection.

Twelve guinea-pigs were inoculated: six were inoculated by shaving the abdomen, then a blister was raised with a heated glass rod: the blister when raised was drained of fluid, and the raw surface so exposed smeared with the flies' fæces containing the bacilli.

Four of the remainder were inoculated intraperitoneally with the centrifugalised deposit from the saline washings already described.

The remaining two were tried by means of the pocket method in the groin.

After two months none of the inoculated animals showed any signs of infection either superficially or on post-mortem examination. This, however, may be due probably to the guinea-pig being an unsuitable animal. (The common white rat of the laboratory is practically unobtainable in this Colony.) It may be that although the lepra bacillus is able to retain its morphology and staining reaction during its passage through the flies' intestines, it loses its infectivity.

In any case, the best prophylaxis is a rigorous campaign against the possible breeding-places of flies: and fortunately as leper asylums, especially in the Tropics, are usually surrounded by a considerable extent of ground, it is more than probable that most of the flies are home-grown and their breeding-places capable of being sought for and destroyed.

NOTES ON THE EXAMINATION OF RATS FOR PLAGUE.

BY H. B. NEWHAM.

In view of the fact that plague has now become a fairly widespread disease amongst human beings, and tends to break out from time to time in communities where least expected, and also as rats, as a general rule, are the first animals to show signs of the disease, it has become increasingly imperative that systematic examination of these rodents for the detection of the disease should form part of our hygienic armament, especially in the case of our large seaport towns.

The manifestations of plague in these animals, as given by various authorities, are:—Buboes, usually in the inguinal or axillary region; enlargement and congestion of the spleen, which may be studded over with small yellowish nodules consisting of necrotic tissue containing masses of plague bacilli: and enlargement and hæmorrhage into the inguinal and axillary lymphatic glands. The bacilli are found in the spleen, liver, lymphatic glands, lungs, buboes, blood, &c.

Thus Hewlett * mentions as evidence of plague in the rat, "Enlargement and congestion of the spleen, and enlargement of and hæmorrhages into the inguinal and axillary lymphatic glands . . . . the spleen may be enormous, six times its natural size, and studded with small yellowish nodules resembling miliary tubercles, consisting of necrotic areas with masses of bacilli; the lungs also may be more or less inflamed and contain small and large necrotic

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* 'Manual of Bacteriology,' 3rd edition.

foci, whilst the bacilli are extremely numerous in the glands and in the spleen, less so in the peritoneal fluid, liver, and blood."

Muir and Ritchie*, describing the appearances of plague-infected rats, say :—" Post-mortem, the chief changes, in addition to the glandular enlargements, are congestion of internal organs, sometimes with hæmorrhages, and enlargement of the spleen : the bacilli are numerous in the lymphatic glands and usually in the spleen, and also, though in somewhat less degree, throughout the blood."

It is evident, therefore, that the spleen is practically a constant site of infection. During the past year I have been engaged in examining some 1000 rats caught or found dead in or near the London Docks, or on board ship, and my method has been to lay open the rats, exposing to view the thoracic and abdominal organs, to lift up the spleen and excise a portion of it, subsequently smearing the cut surface of the same on a slide. This smear is then stained with Löffler's blue or eosin azur, and examined.

In seven instances I have been able to determine the presence of organisms in the spleen which, in morphological characters and staining reactions, were identical with *Bacillus pestis*, and which on further cultural and inoculation experiments proved to be undoubted plague bacilli. In no instance, however, have I ever seen in these infected rats any of the gross macroscopic lesions which by themselves would at once lead me to suspect plague. No hæmorrhage into or enlargement of lymphatic glands was to be noted, no buboes were present, the lungs were not obviously affected, nor were the spleens particularly congested or enlarged or showing necrotic areas. Incidentally it should be remembered that the sizes of spleens, even in healthy rats, vary enormously, so that apparent increase in size does not count for much.

In none of these infected rats were there any signs, therefore, which would lead one, without a microscopic examination, to suspect the presence of plague, though the cultures of the organisms subsequently made from the spleen and other organs when inoculated into susceptible animals proved the bacilli to be highly virulent.

It appears evident, therefore, that plague can exist in rats without giving rise to any gross lesions, and if reliance is placed alone on the signs which are given as the usual evidence of plague in these animals without recourse to microscopic examination mistakes may occur, and some rats may be declared not infected which in reality harbour the plague bacilli in considerable numbers.

It should also be borne in mind that all bipolar staining organisms, even those which may be obtained from the spleen, are not necessarily those of plague. It is absolutely necessary, therefore, that the diagnosis should not rest on morphological and staining characters only, but recourse be had to cultural characters and experimental inoculations. A good instance of this was furnished in the case of one of the rats examined by me.

On opening the rat and exposing the spleen and other organs, no signs were seen suggesting that the animal was infected with plague. On making a smear of the spleen, large numbers of organisms of a bipolar staining character, and in size and shape practically identical with *B. pestis*, were seen. These organisms also were of a remarkably pleomorphic character, thus adding to the probability of the infection being one of plague. Cultures were made on agar &c., and these primary growths were practically indistinguishable from those produced by *Bacillus pestis*. Subsequent subcultures, however, grew less readily.

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* 'Manual of Bacteriology,' 4th edition.

Through the courtesy of Professor Hewlett, who kindly worked out these cultures for me, I am enabled to give the chief cultural and other characters obtained:—

Motility, +.
Gram, +.
Spores, nil.
Agar slope.—Thin delicate granular growth. Growth takes place slowly.
Gelatine slope.—Thin granular growth, only seen after several days: no liquefaction.
Sugar media.—No acid or gas produced.
Broth.—Very faint growth, not apparent for some days.
Blood serum.—Slight growth: no liquefaction.
Potato.—No growth.
Indol.—None formed.
Inoculation.—Two inoculations into rats, subcutaneously and intraperitoneally, produced no effects. One inoculation into guinea-pig, intraperitoneally, produced no effect.

Although at first sight this organism seemed to be the *Bacillus pestis*, it was obvious, especially from its motility, its strong gram-staining property, and its harmlessness to experimental animals, that we had to deal with a different organism. I note that some observers state that the plague bacillus may occasionally be Gram positive. It is possible that these workers may have been confusing the organism I have described, or a closely allied one, with the true *Bacillus pestis*.

LEUCOCYTIC VARIATION IN TRYPANOSOMIASIS.

By H. B. NEWHAM.

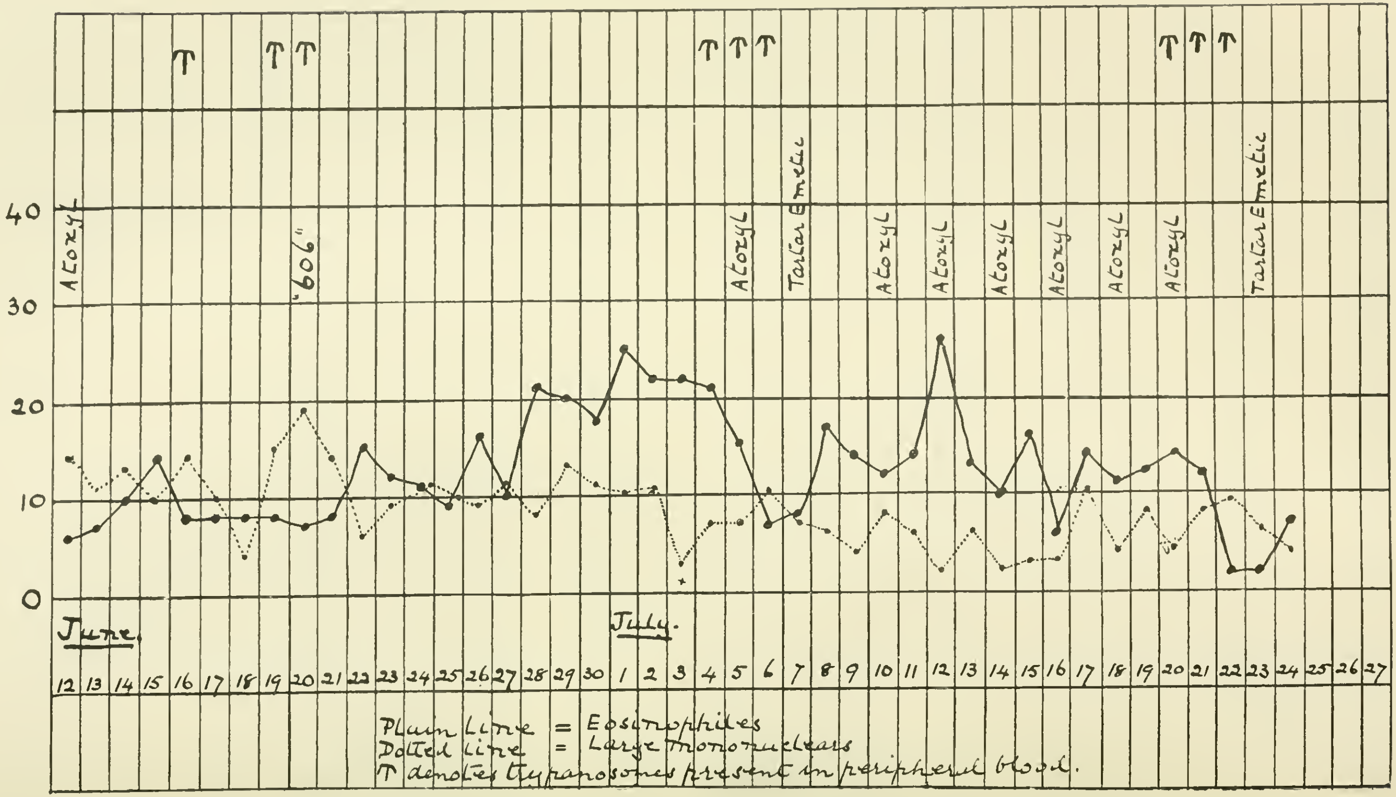
Careful enumeration of the total number of leucocytes and of the percentages of each kind of white cell present has been carried out on many occasions in cases of human trypanosomiasis: and it is generally accepted that in cases of this disease, when fully established, the total number of leucocytes per cubic millimetre is decreased to the extent of some 3000 to 4000 below the normal, whilst differential counts show that the mononuclear elements, especially the lymphocytes, are relatively increased mainly at the expense of the polymorphonuclears.

In the Report of the French Commission on Sleeping Sickness in the French Congo, 1906–8, the results of a series of differential counts of leucocytes on thirty-five patients are given as follows:—

Polymorphonuclears	49·01	per cent.
Lymphocytes	36·60	,,
Large mononuclears	6·36	,,
Eosinophiles	6·24	,,
Transitional forms	0·76	,,

The large mononuclears, therefore, show little modification. The polymorphonuclears are greatly diminished, whilst the lymphocytes show a high

CHART I.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June.
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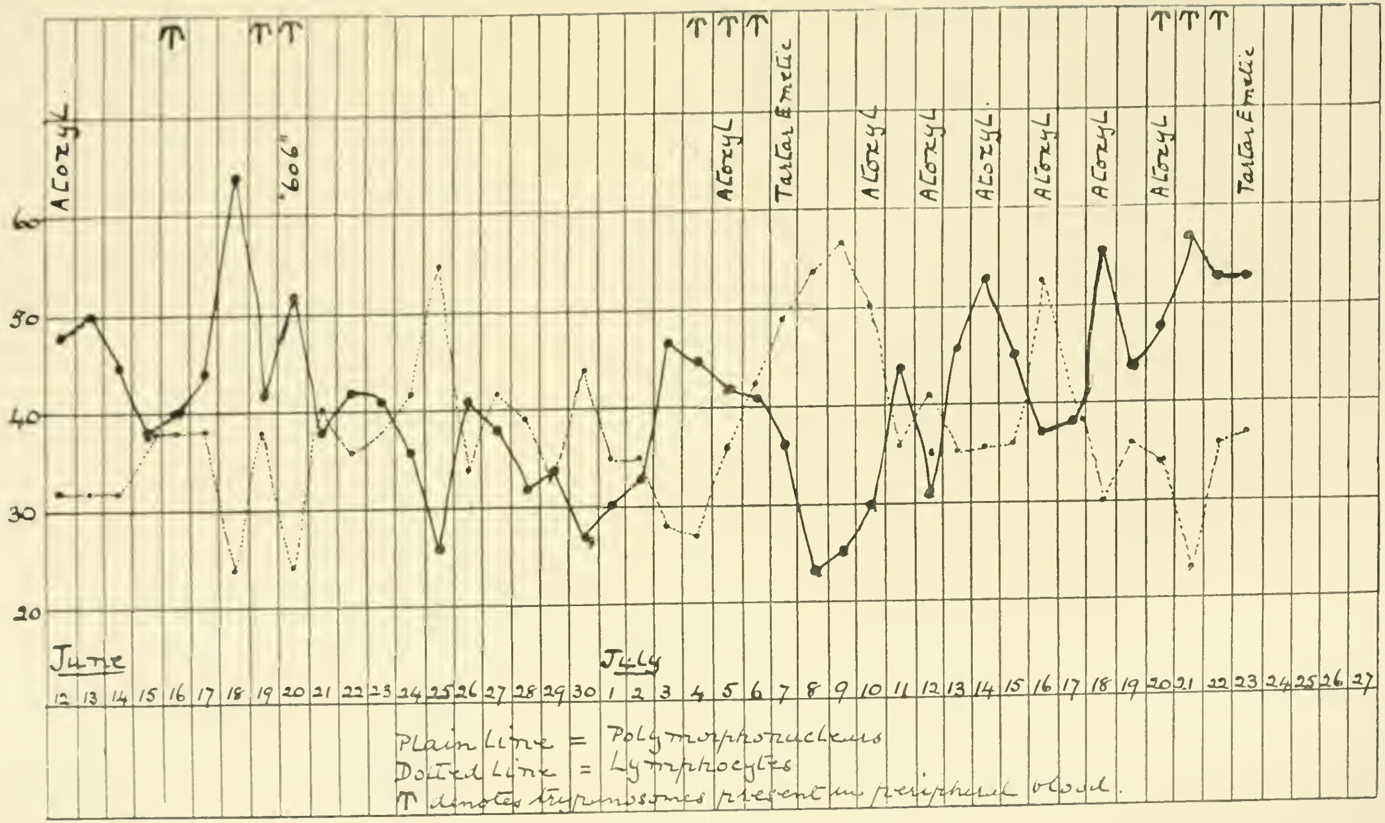
Plain Line = Eosinophiles
Dotted Line = Large mononuclears
T denotes trypanosomes present in peripheral blood.

CHART II.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12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27
Plain Line = Polymorphonucleurs
Dotted Line = Lymphocytes
T denotes trypanosomes present in peripheral blood.

figure. The percentage of eosinophiles is also somewhat high, but is explained by the authors as being due to accompanying filariasis. The figures given appear to be based on single counts, and are not arrived at from a continuous series of counts of each patient.

Observations on the blood of infected animals give much the same results. Yakimoff, working on dogs infected with *T. brucei,* found that from the time of inoculation until trypanosomes could be detected in the peripheral circulation there was a marked leucocytosis, but that this increase rapidly disappeared, giving place to a true leucopenia. This initial leucocytosis was mainly due to a large increase in the polymorphonuclear cells, but that with the onset of the leucopenia these were much reduced, with, at the same time, somewhat of an increase in the mononuclear cells, more especially the lymphocytes. No mention is made of any relative increase in the eosinophile cells.

With the object of ascertaining whether any variation in the relative proportions of the different leucocytes took place when observation extended over some weeks, and also whether the administration of drugs produced any marked effect on the leucocytic count, I have made a series of daily differential counts on the blood of a case of trypanosomiasis at present under treatment in the Seamen's Hospital attached to the London School of Tropical Medicine.

The patient is a young man (34), a big-game hunter by profession, who apparently contracted the disease in North-Eastern Rhodesia, and who has been under observation and treatment for some eighteen months. He has been treated with atoxyl, "606," and tartar emetic (both by mouth and intravenously). The observations extended over about six weeks, a daily differential count of at least 300 leucocytes being done.

On his admission in February 1910, the proportions of leucocytes in his blood were as follows:—

Polymorphonuclears	50	per cent.
Large mononuclears	20	,,
Lymphocytes	20	,,
Eosinophiles	10	,,

It will be noted that this count differs somewhat from the counts quoted for trypanosomiasis, but in our experience there is no constant type. In the first place, the large mononuclear figure is distinctly high, whilst the one for lymphocytes is much below the average in these cases.

On the other hand, one will note that the proportion of eosinophile cells is distinctly large. No cause, such as the presence of intestinal worms &c., could be found to account for this eosinophilia, and one is forced to the conclusion that it was due in some way to the trypanosomiasis.

I am not aware that such a high degree of eosinophilia without obvious cause has been hitherto recorded in a case of trypanosomiasis, except in one case studied by Kerr, who recorded a percentage of eosinophiles varying between 8 and 12 per cent., whereas in the case I have studied the eosinophile figure at one time rose to 26 per cent. Kerr distinctly adds that no intestinal parasites were present in his case. He does not state where his patient contracted the disease.

In the study of the particular trypanosome involved in the case on which my observations were made, it does not vary morphologically from the ordinary *T. gambiense*, but in its behaviour in inoculated animals in some respects approximates to the trypanosome described by Stephens under the name of *T. rhodesiense*.

Possibly, therefore, our patient's trypanosome may be of a different species to the ordinary *T. gambiense*, and produce a different blood-count to that usually associated with infections with *T. gambiense*.

I have endeavoured in the accompanying charts (pp. 38–39) to show the variations in the proportions of the different leucocytes during the period under observation.

In the case of the lymphocytes, large mononuclears, and polymorphonuclears, no particular variation in their proportions could be associated either with the appearance of parasites or the administration of the various drugs.

With regard to the eosinophiles, however, which at one time attained the high figure of 26 per cent., there seemed to be a marked connection between them and the parasites.

With the reappearance of the parasites, a distinct fall in the relative proportion of eosinophiles is well shown, and with the temporary banishment of the trypanosomes from the peripheral circulation by the exhibition of some trypanocidal drug the figure for the eosinophiles is seen to rise at once.

In the periods when the patient was free of demonstrable trypanosomes, the eosinophile figure remained high, and was apparently entirely unaffected by any of the drugs then administered.

It seems therefore, in this case, that there was a distinct interaction between the invading parasites and the eosinophiles; but whether this interaction is a constant one in all cases of human trypanosomiasis, can only be determined by extended observations on a large number of cases of that disease.

TREATMENT OF TRYPANOSOME INFECTIONS.

[M. TSUZUKI.—Zeitschrift für Hygiene und Infektionskrankheiten, vol. lxviii. May 1911.

THE author firstly gives an historical account of the treatment of trypanosome infections. He notes that in spite of the large number of preparations used for treatment, in comparatively few cases has a permanent cure been attained by the administration of one drug alone. Combinations of two or more trypanocidal drugs give better results, but he insists that care must be taken in selecting what combinations to employ.

Experiments on animals infected with Nagana were carried out by the author in order to ascertain what combination of remedies was the most effectual. A large number of substances were tried, but out of them all the only ones which seemed to be definitely of use were arsenious acid, atoxyl, fuchsin, arsenophenylglycin, trypanblue, and tartar emetic.

Various combinations of these substances were used on the experimental animals, and finally it was found that the combination of arsenophenylglycin, tartar emetic, and trypanblue gave the best results. A further point brought out was that by using combinations of these drugs much smaller doses proved effective than if the substances are given singly.

As a result of his experiments, Tsuzuki draws the following conclusions:—

(1) "In the treatment of experimental Nagana the combination of several therapeutically-active substances acts better and produces a surer curative effect in respect to *Therapia sterilisans magna* than do larger doses of the individual components.

(2) "The combination of remedies from one and the same chemical group gives less favourable results (or curative effects) than that from different groups which are chemically less closely related.

(3) "The combination of several substances with different points of attack, in the sense of Ehrlich's law of distribution of medicaments and poisons, as well as after the analogy of the action of narcotics according to Burgi, enables us to produce sterilising mixtures which are relatively non-poisonous to the organism as compared with the toxicity of the sterilising dose of the individual components.

(4) "The best results are obtained by the combination of at least three remedies.

(5) "For the production of more certain curative effects in the Nagana of mice, a second employment of the combined remedies is indicated.

(6) "The question, which remedies are suitable for combination for the purpose of increasing the effect, can be decided for the different species of animals and the different infections only by experiments on infected animals."

H. B. N.

Histological Studies on Egyptian Mummies.

By MARC ARMAND RUFFER.

[Mémoires présentés à l'Institut Egyptien, tome vi. fascicule 3, le Caire, mars 1911.]

Dr. Ruffer reports the results of his work on the "restoration" of the tissues of mummies and dried bodies and their microscopical examination.

The diseases of ancient Egyptians have hitherto been studied by several different methods. Philologists have translated some Egyptian papyri, *e. g.* the "Papyrus Ebers," relating to medical subjects, thereby demonstrating the existence of a medical literature and an extensive pharmacopœia at an early period; but besides giving information of the occurrence of intestinal worms and diseases of various organs, the descriptions of symptoms are too crude to warrant indisputable diagnosis of any one disease.

Professor Flinders Petrie's discovery of veterinary papyri shows that the old Egyptians cultivated veterinary as well as human medicine. Then there are the pictures and statues recovered from temples and some of the oldest tombs. Dwarfs, club-foot, steatopygia, rickets, hunchbacks, cutaneous cysts, have all been figured.

Evidence from historical works of comparatively modern times is of little service; the symptoms noted being indicative not of one disease, but of several.

Information may be gained by the inspection of skeletons, though necessarily confined to those diseases which cause osseous lesions. Professor Eliot-Smith, Dr. Wood-Jones, and Dr. Derry have given valuable descriptions of bones found in the Nile Valley above Assouan.

Although in the process of drying the soft parts shrank and became so changed that pathological lesions are unrecognizable, nevertheless macroscopical examination of mummified organs may give noteworthy results; thus Prof. Eliot-Smith has shown Dr. Ruffer a gall-bladder which undoubtedly contained biliary calculi.

There remains the method of microscopical examination of the tissues of mummies—a method never before systematically applied, though Prof. Looss had demonstrated to his colleagues the striation of mummified muscles.

To the histologist the material appears unpromising. It is very difficult to say to what organ belong some of the brownish dry and hard fragments found in the body-cavity, provisional diagnoses often proving incorrect. It was necessary, therefore, to find a process by which the flexibility and original shape of the mummified tissues could be restored, as affording a hope that in this way recovery, at least partial, of their microscopical structure would be obtained, and that then pathological alterations might possibly be demonstrated. After many experiments extending over two years, such a process was found. The mummified tissues are treated in a solution of alcohol, water, and carbonate of soda, the proportions varying according to the size and consistency of the material to be treated. In some cases 1 per cent. formol was used instead of water and alcohol. The process is delicate, empirical, and requires constant watching. After this treatment the tissue is plunged in chloroform: then follows imbedding in paraffin, and the material is ready for cutting. Microscopic examination gave very satisfactory results. The anatomical elements forming the tissues and organs of mummies and dried bodies being well preserved, the chief microscopical characteristics of skin, breast, lungs, liver, kidneys, testicles, heart, intestines, stomach, blood-vessels, and muscles were easily recognised.

After long histological investigation, Dr. Ruffer thinks that the microscopical examination of mummies may reveal changes due to infiltration of tissues by (1) new growths, (2) infective granulomata, (3) animal and vegetable parasites, (4) inflammation, (5) proliferation of connective tissue (cirrhosis), (6) atheroma and calcification: but that there is little hope of recognizing diseases in which the chief lesions are seen in the cells of organs and tissues.

Dr. Ruffer, after the examination of many hundred bodies, failed to demonstrate red blood-corpuscles or to obtain by any known tests the reaction of haemoglobin from the tissues of mummies or their bandages, in this confirming Prof. Schmidt's former investigations. H. M. H.

V.—BACTERIOLOGY AND PUBLIC HEALTH.

By R. TURNER HEWLETT, M.D., F.R.C.P., D.P.H.

Acid-fast and Acid-resisting Germs cultivated from Cases of Human Leprosy, and their Determination.

By H. BAYON, M.D.,
Craggs Investigator, London School of Tropical Medicine.

The following experiments were carried out with the view of answering this set of questions in connection with Leprosy, viz.:—

(1) Can the germ of leprosy be cultivated from the lesions it causes?

(2) Can leprosy be transmitted to animals either direct from the human being or after cultivating the germ?

(3) What is the explanation of previous unsuccessful attempts?

(4) What methods can be made use of to determine the relationship of germs cultivated from cases of leprosy to the disease itself?

(5) Is rat leprosy caused by a germ identical with that of human leprosy?

(6) How is leprosy transmitted? Is there any evidence of its being carried by arthropods?

(7) What lines of treatment can be recommended, in view of the answers obtained to these questions?

Acid-fast and acid-resisting germs are so ubiquitous, and bacteria of the smegma group have been found in so many different parts of the body, that it is certainly not an excess of prudence to thoroughly test any germ cultivated from cases of leprosy as to its relationship to the disease, without relying too much on the morphology and staining properties of the bacteria obtained. Unluckily, acid-fast organisms do not give always clear results by means of serological tests, and animal experiments are not always as conclusive as one might wish.

On undertaking, therefore, these investigations for the London School of Tropical Medicine, I decided not to make any publication of results till I had been able to apply all possible methods of identification to the germs I might obtain from cases of human leprosy.

Some of my results have already been embodied in a paper I read before the Tropical Diseases Section of the British Medical Association at Birmingham this year.

A glance at the appended list of authors (see p. 16) who have published the results of their attempts to cultivate the germ of leprosy shows that, time after

LIST OF PUBLISHED SUCCESSFUL ATTEMPTS TO CULTIVATE THE *BACILLUS LEPRÆ*.

NAME OF AUTHOR.	DATE.	MORPHOLOGY OF GERM.	STAINING PROPERTIES.	REMARKS.
Bordoni-Uffreduzzi	1888	Diphtheroid.	Acid-fast.	
Babes	1889	,,		
Gianturco	1889	,,	Acid-resisting.	
Campana	1891	?	?	Anærobic.
Kanthack	1891	?	Acid-resisting.	
Ducrey	1892	Same as	Campana.	
E. Levy	1897	Streptothrix.	Acid-resisting.	
Sticker & Dieudonné ..	1897	Rods.	Acid-fast.	
Czaplewsky	1898	Diphtheroid.	Acid-resisting.	
Teich	1898	?	?	
Carrasquilla	1899	?	?	
Barannikow	1899	Diphtheroid.	Acid-resisting.	
Kedrowsky	1901	,,	,,	
F. Levy	1901	,,	,,	
Van Houtun	1902	?	?	Gram negative !
Klitin	1903	Diphtheroid.	Acid-resisting.	
Karlinski	1903	,,	,,	
Zenoni...............	1904	,,	,,	Admitted to be contamination.
Rost	1904	?	?	
Deycke & Reschad	1905	Diphtheroid.	Varying.	
Weill	1905	Rod.	Acid-fast.	Culture died out.
Shiga	1909	Diphtheroid.	Acid-resisting.	
Clegg	1909	?	?	
Duval	1910	Short rods.	Acid-fast.	Similar to *B. phlei.*
Rost	1910	Streptothrix.		
Williams.............	1910	,,		
Twort	1910	Rod.	Acid-fast.	Culture died out.
Bayon	1911	Diphtheroid and rod.	Acid-resisting and acid-fast.	

Note.—I call all germs simply "acid-resisting" that do not get bleached by 2 per cent. sulphuric acid in five seconds after having been stained with warm carbol fuchsine for ten minutes.

"Acid-fast germs" withstand treatment with 20 per cent. nitric acid for thirty seconds or more after the same procedure. Both acid-resisting and acid-fast germs should not take Löffler's blue after bleaching, but remain red.

time, a diphtheroid appears to be the result of their labours, and in fact it is found to the credit of the best-known workers in the field. And yet up to this very day none of these cultures has succeeded in achieving general recognition, probably because, except in the case of Kedrowsky, no author appears to have been able to produce in animals, with the germs he cultivated, anything similar to the lesions seen in leprosy of the human being.

From the nodules of a case placed at my disposal at the London School of Tropical Medicine by Sir Malcom Morris, I succeeded in cultivating on two occasions an acid-resisting diphtheroid. I had also been able to isolate the same germ from the nodules of another leper at the Seamen's Hospital, and had allowed it to die out, because I did not recognize its importance at the time.

Moreover, I have cultivated on nutrose agar horse serum (with the addition of 2 per cent. ground-up smegma bacilli, according to a variation of Twort's formula) an acid-fast rod, morphologically identical with the bacilli seen in culture nodules. It is so far mixed with the diphtheroid and is at its third subculture, and has taken five months to grow.

This diphtheroid I injected into rats and mice and regained it by culture and in sections as an acid-fast rod, which, however, still has a certain resemblance to a diphtheroid. This culture is practically identical with the one Kedrowsky obtained from two cases of leprosy at Moscow.

I also cultivated a streptothrix, which did not resist the action of acids in the least, and which broke up into acid-fast rods of a diphtheroid character on injection into rats.

A streptothrix was regained by culture by my assistant, Dr. Martinez, from a rat injected with Kedrowsky's germ.

These results seem to confirm the experimental work of Kedrowsky, who considers the lepra "bacillus" to be the fragments of a streptothrix, and who has brought an extensive series of careful experiments to prove his view.

Beauchamp Williams, working quite independently of Kedrowsky, in Burmah, has come to the following conclusions :—

". . . That leprosy may be caused perhaps by several strains of an extremely pleomorphic streptothrix, which under certain circumstances may be :

"(1) A non-acid-fast streptothrix with interlacing filaments ;
"(2) A non-acid-fast diphtheroid bacillus, which is in reality a streptothrix and capable of becoming acid-fast under certain defined conditions ;
"(3) A definite acid-fast filamentous streptothrix ; and
"(4) An acid-fast bacillus, which is the broken-down stage of a streptothrix."

To this evidence we may add the results of Sanfelice, who, in injecting into dogs and guinea-pigs various strains of *Streptothrix alba* and *violacea*, found them to break up into acid-fast rods indistinguishable from tubercle bacilli.

Moreover, Birt and Leishmann have published a case of human streptothricosis of the lungs, with no evidence of tuberculosis at autopsy, which showed acid-fasts in the pleural exudate.

I have had the greatest difficulty in persuading myself about this astonishing pleomorphism, but have had to give way before the incontrovertible results of my repeated experiments.

Moreover, the literature on the subject is studded with evidence that many, possibly all, acid-fast germs are but the one definite stage of streptothrices, and it only needs careful piecing together to obtain a complete picture of the process involved in this transformation.

An easy cultivable germ that shows a similar pleomorphism is the smegma bacillus, which I cultivated off my own body and I find to be identical with Moeller's strain.

There is therefore no difficulty in explaining previous unsuccessful attempts to cultivate the germ of leprosy. A streptothrix is generally considered to be a contamination from the air, and the tube gets thrown away.

Moreover, even the streptothrix and the diphtheroid stage grow in the first generation with extreme difficulty and only on special media. I have made use of placental agar (prepared according to Kedrowsky) and of nutrose horse serum agar.

As a matter of fact, several observers have no doubt cultivated the leprosy germ, but have not succeeded in making good their claim owing to manifold and persistent difficulties encountered in the methods for identification.

Even if the cultures are injected into rabbits or guinea-pigs, the resulting lesions are not visible to the naked eye, and under the microscope are easily overlooked.

The proper animal to use is the rat: the preferable site for injection is the tunica of the testicles. In this way I got positive results, which cannot be distinguished from the genuine or spontaneous leprosy in rats and are completely analogous to the lesions found in man.

Once the strain is set going as an acid-fast rod on placental agar, it can be easily subcultured on the various media used for tuberculosis, such as glycerine agar, glycerine potato, &c. However, I am not laying stress on the germ obtained, but on the methods used to identify it. That is the paramount difficulty in the whole question.

Given an appropriate animal, the rat, and given an appropriate method of injection, *i. e.*, into the tunica of the testes, leprosy appears to be transmissible to animals; therefore proving its contagiousness.

Even here successful results have often been reported, but not admitted owing to the results of the experiments of Campana, Leloir, Wesener, Huppe, and others, who found that pieces of tissue with leprosy bacilli in them—leprous tissue—which had been in alcohol or dried for months, when implanted into animals gave rise to lesions similar to those described by those authors who claimed to have transmitted leprosy to animals direct from the human being.

These experiments I have repeated. I injected autoclaved cultures of smegma and Kedrowsky's germ into the peritoneum of guinea-pigs, and intravenously into rabbits. Extensive macroscopical lesions resulted; on autopsy the organs showed germs in enormous quantities, in great quantities in liver and spleen; all attempts at culture failed. Similar experiments have been made with tubercle bacilli. The whole group of acid-fast germs seems to be specially resistant to the lytic properties of the animals, and gets eliminated but slowly. I suppose this is partly due to the waxy substance which envelops them, forms their outer coating, and which seems to be as quiescent as paraffin is in tissues. Accordingly but slight reaction is set up, and under the circumstances the only difference in behaviour between dead and living germs is that the latter may increase in numbers, and commonly do so, whilst the former get simply eliminated after longer periods of quiescent permanence.

As a control I regained on several occasions the living acid-fast germs

I had injected, though the lesions were in most cases less marked macroscopically than those caused by dead germs.

This point settled, I had to find out whether the germ I had cultivated out of the lesions produced in the rat was not one of the commonplace known acid-fast germs. For this purpose I compared simultaneous cultures on the following media :—

> Glycerine gelatine, glycerine agar, glycerine potato, Dorset's egg-medium, nutrose horse serum agar, placental agar.

The strains made use of were the following :—

> Human, bovine, and avian tuberculosis from the Lister Institute. One smegma strain from Moscow, one I cultivated myself, and one from Kral. Timothy grass, Moeller's, von Gras, and Grasberger from the Lister Institute. Tobler one, three, four, and five, and a Rabinowitsch from King's College. Arloing-Courmont's homogeneous tuberculosis, Marpmann's Harn bacillus, Moeller's smegma, Karlinski's acid-fast from the nasal mucus : all obtained from Kral.

The rate of growth soon eliminated all these germs but the tubercle group, to which I must add Johne's pseudotuberculosis, a culture of which was kindly given me by Dr. Twort, of the Brown Institution.

The difference in appearance of all these germs, excepting, again, the tuberculosis group, was also very marked when compared with Kedrowsky's germ and the germ I had cultivated from my case. The nearest approach was the smegma bacillus, and I thereupon carried out the following experiment :—

A rabbit was injected intravenously with 2 cub. cm. of thick emulsion of living smegma bacilli. After three weeks the rabbit developed nodules in the skin, which broke down and showed acid-fast germs in the pus. The breasts got inflamed and swollen. The animal became thin, and its left hind leg went lame.

We had here a complete analogue of a case of leprosy in the human being, clinically a mixed case, nodules and nervous lesions.

The animal was killed. Acid-fasts were found in enormous quantities in the breasts; the kidneys were honeycombed with caseous nodules. The microscopical appearances were distinctly different from anything seen in leprosy, and reminded one much more of actinomycosis in parts.

Even from the blood of the heart I succeeded in regaining the germ in this case.

The experiment was repeated with identical results.

The question remained, Had I not sown leprosy and reaped tuberculosis ?

I therefore injected three guinea-pigs with heavy doses of the germ cultivated from cases of human leprosy. Two were killed after four and six weeks ; no signs of tuberculosis, though acid-fasts were found in the spleen. The other animal is still living, alive and healthy, after six months.

The sections made in no case warranted the diagnosis human or bovine tuberculosis.

As regards avian tuberculosis, I injected two pigeons with equal quantities of avian tubercle culture and lepra germ culture. The control died of avian tubercle ; the other is still alive and well.

I had to carry out this experiment, because the intravenous injection of avian tubercle in the rabbit gave rise to appearances very similar to those obtained by injecting the lepra germ. The differentiation has, however, also been carried out by means of serological methods.

The animal experiments are still being continued; but in the meanwhile it was necessary to find methods capable of proving the relationship of the germ I had cultivated to the disease it caused. The two methods most generally used for this purpose are agglutination and complement deviation. For tuberculosis Koch had devised the precipitation test.

A cursory glance through the literature on the subject shows, however, that as far as tubercle is concerned, all these methods have been unable to be satisfactorily applied. This is due again to the waxy substance surrounding the bacteria of the acid-fast group, which renders the preparation of a satisfactory antigen a matter of great difficulty, and possibly, moreover, that antibodies are formed in a very incomplete fashion in the case of tuberculosis.

However, I decided to make use of these methods, and after a few initial experiments on animals I began to attempt to produce a satisfactory antigen. After numerous unsuccessful attempts, the following method was found to be the best:—

One part by weight of moist culture is suspended in ten parts of distilled water. The tube is sealed and shaken for half an hour in a Hearson's shaker. After that it is frozen for twelve hours at least, and then rapidly thawed with the intention of splitting up the bacilli. This process is repeated three times, and after each thawing the tube is thoroughly shaken. In conclusion, it is heated twelve hours at 60°.

The serum of the case Giraud was taken, inactivated, and the following tests were carried out:—

I. Serum diluted from 1/1 to 1/256. Antigen 0·5 c.cm. in each tube. One hour at 37°.

Amboceptor (double dose) and 0·5 sheep's blood suspension (1/20).
No hæmolysis after two hours at 37° in all tubes from 1/1 to 1/64.
Hæmolysis begins at 1/256.
Controls.—Antigen, serum for auto-inhibition, serum for complement: amboceptor and complement.

II. Serum of a syphilitic patient who gave a positive Wassermann reaction (given me by Dr. Petrie).

Negative in all dilutions with same antigen.
Controls same as above.

III. Serum of another syphilitic patient from St. George's Hospital.

Same result as above.

Leprosy serum tried simultaneously against lepra and avian antigen. Serum diluted from 1/5 to 1/320. Antigen used in full. 1/2, 1/4, and 1/8 doses against all these dilutions.

Controls as before.
Result.—Leprosy antigen:
No hæmolysis up to 1/80 against all dilutions of antigen.
Avian antigen:
No hæmolysis up to 1/20 against full doses of antigen.
,, ,, 1/10 ,, 1/2 ,, ,,
,, ,, 1/8 ,, 1/4 ,, ,,
Hæmolysis present, 1/5 ,, 1/8 ,, ,,

Leprosy serum tried against smegma antigen.

Hæmolysis began at 1/32.

Leprosy serum tried against Timothy grass.

Hæmolysis began at 1/10.

I believe these results may claim to be highly satisfactory, though so far they do not admit of generalization.

Sir Arthur Downe, of the Local Government Board, very kindly helped to collect three sera from cases of leprosy and alleged leprosy.

One nodular case who has had prolonged Nastin treatment reacted one in ten to the antigen.

A nervous case of many years' standing reacted one in five.

The alleged case gave a frankly negative reaction in all dilutions.

I got these last results a short time ago, and have repeated them with new antigen.

The importance of these tests is not so much in their application to the diagnosis of the disease, great as possibly may be their usefulness in this line, but in the possibility of controlling the results of treatment by means of specific bacterial products.

We have seen that the lepra bacilli, in common apparently with other acid-fasts, have the property of causing very similar lesions irrespective of the fact whether they are dead or alive.

This fact must be kept in view during vaccine treatment. We might cause a nodule at the site of injection, and consider it as a result of having injected living germs. We have also seen how resistant dead germs are, and how they can remain in the body for a long time without causing any marked reaction. Therefore any vaccine treatment undertaken must have broken-up dissolved bacilli as a basis.

Following up some of Much's and Hoessli's work, I have found that smegma bacilli can be completely emulsified by 1 per cent. citric acid in fourteen days. The acid can then be neutralised to sodium citrate and the total emulsion injected.

I am applying these results to the lepra germ, but do not intend to begin treatment till after further experiments on animals.

On the other hand, I have prepared a bacterial culture extract made exactly on the lines of alt-tuberculine. This has been injected into the case I have in treatment at St. George's Hospital. It gives a marked reaction: nodules in the legs appear to have melted down, and the man says he feels much better. I wish to be very reticent on the subject: so many treatments appear, are extensively advertised, and then fizzle out, that I do not wish to make any claims that I cannot substantiate on several cases.

I have begun immunising a goat, and intend also using its serum as a method of treatment.

It is to be understood that no treatment will be able to have the effect of quinine on malaria. No bacterial disease with such a chronic course as leprosy can be healed in a twinkling. But I believe we shall be able to arrest the multiplication of the bacilli, cure open sores, prevent new ones forming, and at the very least keep the disease quiescent for an unlimited time. All this cannot be guaranteed so far.

I also injected an extract of the culture of the smegma bacterium; and though its concentration was about fifty times stronger than the lepra extract, it caused no reaction when injected into the patient.

We need not waste time considering the fish hypothesis, and whether leprosy is hereditary. These are subjects about which nothing new can be said.

On the other hand, we know that though leprosy is contagious, it is so to such a slight degree that its infectiousness has repeatedly been denied.

My experiments have led me up to the conclusion that the acid-fast germ seen in the nodules of lepers is infective to a very slight degree. To achieve infection it has to be injected in relatively big quantities. This appears not to be the case with the diphtheroid stage. Here, however, the incubation can be very long if the germ is not injected in sufficient numbers.

Before exact and complete experiments can be carried out with arthropods, we must know more about the non-acid-fast stages of the germ.

Mosquitoes apparently do not play a rôle, and flies eliminate acid-fasts within two days. Bed-bugs have lately been incriminated ; but here, again, only the acid-fast stage of the germ has been considered.

What are the facts available on this subject?

That only very intimate contact, so intimate as not to exclude direct mechanical infection, seems to produce leprosy in a certain number of cases. In others again, though the people have been a short time in leprous country, they have not even seen, to their knowledge, cases of lepra. In these cases a transmission by arthropods is admissible—even highly probable. But here, again, the primary lesion seems to be about the face, especially the lobes of the ears, the alæ of the nose, the forehead, and the chin ; later on, the eruption may appear on the limbs.

There is no arthropod we know of that prefers to bite about the face. Mosquitoes bite about the legs, and also often, I admit, about the ears. Fleas more frequently on the trunk and legs. Simulids (at least the *Simulium damnosum* in Uganda) hands and forehead, where they appear to be attracted by the moisture of perspiration. Bugs and body-lice about the trunk. *Phthirius pubis* about the trunk, and sometimes, but very rarely, the face. Tsetse-flies, ticks, &c. are excluded till further proof, because of the wide distribution of leprosy. Our last resource is the *Sarcoptes scabiei* and the *Demodex folliculorum.* Some facts speak for a possibility of their being able to carry the bacilli where they may thrive. The demodex is found actually in the positions where nodular lepra is more apt to break out. Its range is very small.

This is as far as I wish to make any statement before any experiments are concluded.

It should not be forgotten that nervous and tuberous leprosy may be due to different modes of infection. We have an analogue in tuberculosis, and in rats different organs are attacked by the lepra germs according to the way infection takes place. Spontaneous leprosy shows marked skin lesions. Intraperitoneal injection gives rise to deposits in spleen and liver ; subcutaneous or intravenous injection is often followed by pneumonia.

I have only quite lately received some rat leprosy, and am at present studying the relation of this disease to the human variety. I consider that both are due to the same germ, for the following reasons :—

I. Experimental.—Leprosy produced in the rat by injection of cultures from the human being cannot be distinguished histologically from one another.

II. The serum of human lepers reacts up to one in eighty with the germ of spontaneous rat leprosy (*Dean*).

Conclusion.

My results are the following :—

From a case placed at my disposal at the London School of Tropical Medicine, I succeeded in cultivating on two separate occasions an organism which became acid-fast on being injected into mice and rats. I regained the germ in pure culture: succeeded in proving it was not tubercle—human, bovine, and avian—and not smegma. I distinguished it by cultural methods from nineteen other acid-fast organisms. By injecting this germ I produced in rats the identical changes of spontaneous leprosy of the rat and analogous to the lesions of leprosy in the human being.

Leprosy was transmitted directly to the rat from the human being, definitely proving that the disease is contagious.

The complement deviation-test was adapted to the special circumstances of leprosy by preparing an appropriate antigen, and a successful differentiation was made between various germs which might have been mistaken for leprosy, proving at the same time the relationship of the bacterium cultivated to the disease. The method may yet be of great use in diagnosis, and in any case will enable treatment to be controlled.

Promising therapeutic attempts have been begun, and a complete explanation has been given of previous failures to infect animals and cultivate the germ.

The Bacteriological portion of these researches was carried out at the Lister Institute of Preventive Medicine; and I desire to express my indebtedness to Dr. C. J. Martin, F.R.S., and the Staff of the Institute for the help and advice they have so freely given me.

PLAGUE IN EAST ANGLIA.

THE recent appearance of Plague in East Suffolk forms the subject of some valuable reports issued by the Local Government Board*. The reports are divided into three sections, each of which deals with the subject from different aspects; and they are prefaced by an introductory letter addressed to the President of the Local Government Board by Dr. Newsholme, the Principal Medical Officer of the Board. In this letter a brief outline of the course of events is presented.

As a result of a very pains-taking and searching enquiry on the part of the late Dr. Bulstrode (by whose recent death the Medical Department of the Local Government Board has sustained a great loss) it was ascertained that the four cases of human illness which occurred in the latter days of September 1910 had been not only accompanied, but also preceded for some time, by a fatal epizootic among the rats of the locality. This epizootic, continuing after the cessation of the outbreak of human plague, was proved to be plague. Some hares, a cat, and a ferret were also found to have died of the disease. The case of the cat, examined by Professor Simpson, was interesting in that it had been buried and had to be disinterred for examination; yet there was no difficulty in obtaining pure cultures of the bacillus.

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* 'Reports and Papers on suspected cases of Human Plague in East Suffolk, and on an Epizootic of Plague in Rodents.' 'Reports to the Local Government Board on Public Health and Medical Subjects,' New Series, no. 52 (1911)

The distribution of plague infection covered an area of nearly 15 square miles, but was fortunately in a sparsely populated district and one which was isolated. In order, however, to be sure of the extent of country infected, a systematic examination of rats was instituted over a belt of country surrounding the infected area. This investigation was carried out by Drs. Petrie and Macalister. They examined 6071 rats in this belt, with the satisfactory result that none was found to be infected with plague. Every one of the 6071 rats was of the *Mus decumanus* species.

The scientific work on rat-fleas carried out by Drs. C. J. Martin and Sydney Rowland gave some interesting results. The rat-flea of India (*Xenopsylla cheopis*) could not be found on any of the rats examined; more than 50 per cent. of the rat-fleas in East-Sussex consisted of the *Ctenophthalmus agyrtes*, which does not bite man. The remainder of the rat-flea population was composed of *Ceratophyllus fasciatus*, which bites man, but not so readily as does *Xenopsylla cheopis*. It is estimated that, on an average, there is less than half a man-biting flea for a rat. This, Dr. Martin points out, "is an infestation which was not found sufficient to give rise to a human epidemic in India, notwithstanding the vastly greater accessibility of mankind for rat-fleas." This observation is, however, qualified by the remark that the "observations were made in November, after the onset of cold weather, and that at this season of the year the rate of breeding of fleas is at a minimum, if not entirely negligible."

Interesting and valuable as these observations are, too much importance should not be attached to them, even if the rat-flea infestation proves to be small in the summer and early autumn. The four deaths at Freston were of the pneumonic type of plague. The first three occurred in a four-roomed cottage, where daughter, mother, and father in a family of six were attacked by an obscure lung affection which proved fatal in a few days. The other occurred in a cottage a quarter of a mile away, but the victim had nursed the mother and had evidently caught the infection from her patient. The main features of the illness were the obscurity of the symptoms, the dark-stained expectoration, the high temperature, the great prostration, and the rapidly fatal issue in about three days without any corresponding serious physical signs to account for it. Bateriological examination by Dr. Heath, the Honorary Bacteriologist to the Ipswich and East Suffolk Hospital, fortunately cleared up the nature of the disease, which clinically had puzzled the medical men attending the patients. If the cases had not occurred a few miles from the hospital, where a bacteriologist was stationed, probably the disease would have remained undiagnosed, and would have been overlooked in a similar way to that of an outbreak which Dr. Bulstrode's investigations traced as having occurred in December 1909 and January 1910 at Frimley, a village separated from Freston by the River Orwell. Here, in a two-roomed cottage occupied by a family of seven in poor circumstances, the whole of the family was attacked by an illness unfamiliar to the medical men and in some cases associated with glandular enlargements. Four of the family recovered, and three died. Dr. Bulstrode came to the conclusion that the malady was bubonic and septicaemic plague.

Again, in 1906 at Shottley, a village about four miles from Freston, eight persons were attacked with what appeared at the time to be a virulent form of influenza, and six of these died after three to four days' illness. This outbreak had also been preceded by an unusual mortality among rats.

The insidiousness, slowness, and deceptive character of plague, which stand it in good stead in acquiring a lodgment in a healthy country, are well exemplified by the evidence furnished by these reports, and emphasize the need of extraordinary watchful attention.

How the plague infection reached East Anglia was not cleared up; but it is probable that it was imported by plague-infected rats brought in grain-ships from some of the infected ports in South America. Ipswich, which is in the centre of the infected district, though not itself infected, is a large grain emporium.

Ports and Plague Infection.

[Outbreak of Pestis among Rats in Hull. By J. Wright Mason, Medical Officer of Health.]

The risk to which ports, whose trade relations bring to them vessels from infected ports, are exposed is instanced by the occurrence in Hull in February 1909. In one of the sheds at the Dock a number of dead rats were discovered which, on examination, were found by Dr. Wright Mason, the Medical Officer of Health, and also by Dr. Klein, on behalf of the Local Government Board, plague infected. From December 1908 to the end of February 1909, 800 vessels from suspicious and plague-infected districts, viz. Alexandria and Karachi, had discharged cargo on the south side, where the shed was situated. Medical inspection of these vessels had not discovered any illness among passengers or crew. The precautions adopted appear to have stamped out the epizootic.
W. J. S.

Transmission of Plague in the absence of Rats and Rat-Fleas.

[By E. A. Walker, M.B., Capt. I.M.S.—The Indian Medical Gazette, May 1910.]

In an outbreak of plague in the villages of Kyankin and Twather, Meiktila district, Capt. Walker placed the inhabitants in a segregation camp, but cases of plague continued. This being the case, he had collected the bed-bugs from the bedding, clothing, mats, &c. of the segregated villagers, with the result that of bugs collected from infected huts, 6 out of 27, or 22 per cent, were infected with *Bacillus pestis*. Further, taking 5 bugs and placing them on the leg of a bubonic case of plague and allowing them to remain for 10 minutes until they had drawn blood, Capt. Walker then put them on a rat's abdomen, which they bit freely. In 60 hours the rat died and showed in smears of the spleen plague bacilli. Capt. Walker draws attention to Verjbitski experiments conducted in 1902 and 1903, in which he proves the transmission of plague in guinea-pigs by bed-bugs.
W. J. S.

CULTIVATION OF THE LEPROSY BACILLUS.

[Scientific Memoirs of the Government of India, no. 42.—Part 1. The Cultivation of the Bacillus of Leprosy and the Treatment of Cases by means of a Vaccine prepared from the Cultivations, by Major ROST, I.M.S. Part 2. The Cultivation of the Leprosy Bacillus, by Capt. WILLIAMS, I.M.S.]

THE successful cultivation of the leprosy bacillus has been announced time and again by various workers, but their results on being examined have not satisfied the world of science.

Now, however, it seems fairly certain that Major Rost and Capt. Williams have succeeded in cultivating this organism, and, what is more, have obtained hopeful results by means of vaccines in the treatment of several cases of leprosy.

Rost used as his medium a mixture of distilled volatile alkaloid of rotten fish, Lemco broth (without salt or peptone), and milk. Inoculations of this medium with material from a leprotic lesion produced a slight stringy growth at the bottom of the tube, which, on examination, was found to consist of large numbers of acid-fast bacilli massed together in parallel arrangement.

In subcultures on nutrient agar a growth was obtained which was only slightly acid-fast. The acid-fastness, however, was rapidly regained on transferring to a medium of a fatty nature.

In all, Rost was able to obtain cultures from three different cases which all showed very much the same characteristics. Inoculation experiments with the cultures were tried on monkeys, and in one case, after repeated injections extending over some three months had been given, the animal developed symptoms strongly suggesting leprosy. He developed exacerbations of temperature varying between 100° and 103°, and nodules were noticed over the superciliary ridges, on the cheeks and inner aspect of the left ankle, and on the dorsum of the left foot. Examination of the contents of these nodules showed large masses of acid-fast organisms exhibiting parallel arrangement.

Rost next prepared a vaccine from his cultures, and this was tried on 10 lepers who volunteered for the treatment. In all these cases marked improvement is recorded, so much so that two of the ten cases are stated to be cured. Various modifications of the preparation of this vaccine are being tried and a fuller report of the results are promised later.

Williams' results carry us further. Not only has he been able to obtain cultures of acid-fast bacilli similar to those obtained by Rost, but from leprotic lesions he has also been able, with suitable media, to grow an acid-fast streptothrix.

As was suggested by Foulerton in his Milroy lectures of 1910, it is highly probable that a disease may be caused by a streptothrix and yet the obvious parasitic form found may be always a bacillus or some other broken-down streptothrix form.

Williams believes that both the streptothrix and the bacillus isolated by him are practically one and the same thing, the bacillus being simply a broken-down form of the streptothrix. As evidence of this, he finds that the streptothrix growth when grown in milk breaks down into bacillary elements, and he has also succeeded in showing that by suitable cultivation the streptothrix form can be produced from a culture showing only bacillary forms.

Williams finds that he can fairly readily obtain the streptothrix growth in ordinary media such as broth, agar, &c., but for the bacillary type of growth he resorts either to Rost's medium or a modification of the same, in which distilled water replaces the rotten-fish distillate.

His inoculation experiments with both organisms made on monkeys, guinea-pigs, rabbits, and rats have so far proved negative.

Preparations of vaccines from both the streptothrix and bacillary cultures have, when inoculated into lepers, produced marked reaction and some definite improvement in their condition, and it is to be hoped that with further investigation a safe and efficient vaccine may be forthcoming.

H. B. N.

The Existence of Undulant Fever in Portugal.

[Nicolau Bettincourt.—Archivos do Instituto Bacteriologico Camara Pestana, vol. iii., January 1911.]

Undulant fever has for some time been known to have a much wider distribution than its synonyms—Malta or Mediterranean fever—would seem to convey. Not only is it found in practically every country bordering on the Mediterranean, but cases have been reported from such widely separated places as South Africa, India, and, perhaps, West Africa. Hitherto it has not, however, been proved to occur in Portugal, though several physicians there, from clinical symptoms only, have for a considerable time suspected its occurrence.

Bettincourt has now, however, been able to demonstrate by bacteriological methods the existence of this disease in that country. He was not able to secure cultures of the causative organism from either the blood or the spleen of the eight cases he investigated, but resorted to agglutination tests and fixation of complement. These were in all cases positive, and leave little doubt that the cases under consideration were definitely caused by the *Micrococcus melitensis*.

The cases all occurred in individuals who had never been out of the country and clinically presented the usual train of symptoms associated with undulant fever. Six of the cases resided in one small district.

On further enquiry, he was able to satisfy himself, in six out of the eight cases at least, that the origin of infection was in all probability goats' milk.

The author does not record, however, that he made any attempt to investigate the prevalence of the disease among the goats. Such an enquiry would add greatly to our knowledge of the distribution of the disease. H. B. N.

Experimental Yaws in the Monkey and Rabbit.

[Nichols.—Journal of Experimental Medicine, no. 5, 1910.]

Much controversy has in the past raged round the vexed question of the relationship, if any, between Yaws and Syphilis, and numerous investigations and experiments have been undertaken to endeavour to settle the question.

The results obtained by Castellani in 1905 went a long way towards solving the question. Not only did he discover what is believed to be the causative agent of Yaws, the *Treponema* or *Spirochaeta pertenuis*, but he was able to reproduce yaws in monkeys by artificial inoculations, and from a survey of his whole work he came to the conclusion that yaws and syphilis are two entirely separate diseases.

Nichols gives an account of experiments carried out by him which serve still further to strengthen Castellani's conclusions.

It had been shown by Parodi and Uhlenhuth and Mulzer that injection of the *Treponema pallidum* into the testicle of the rabbit produced a syphiloma in that organ and that from the affected organ a rich supply of the causative organism—*Treponema pallidum*—could be obtained.

Bearing these facts in mind, Nichols attempted to infect rabbits with the spirochæte of yaws in the same manner. In these attempts he was successful. From a human case of yaws he successfully inoculated a monkey and from that animal was able to transfer the infection through three generations in rabbits. The incubation period was some forty-one days in the first generation, forty days in the second, and twenty-five days in the third, thus showing a shortening in the third passage. The lesions produced in the testicles were carefully studied, and although they much resembled those produced by syphilis, still points of difference could be made out. The nodules in syphilis were found to be larger and their centres were necrotic, whereas in the yaws lesion the tissue was homogeneous throughout, and the accompanying interstitial orchitis less marked. Serum reactions were also investigated and positive complement fixation reactions were obtained in several cases in the rabbits infected with yaws.

Nichols comes to the conclusion that the surest way of differentiating the organisms is by considering the differences in the lesions produced by both syphilis and yaws in the monkey. He finds that in yaws the incubation period is two to three weeks, the lesion is elevated, slightly scaly, and very œdematous, whereas in syphilis the incubation period is some four weeks and the lesion is flat, dry and scaly. He also considers that slight morphological differences could be detected in the respective causative organisms. These, however, seem to be of such an indefinite nature as to be unreliable.

H. B. N.

THE PREPARATION OF A SAFE AND EFFICIENT ANTIRABIC VACCINE.

[Lieut.-Col. Sir D. SEMPLE.—No. 44 of the Scientific Memoirs of the Government of India.]

AN account is here given of Lieut.-Col. Semple's investigations and experiments for the production of an antirabic vaccine of an efficient and safe nature.

Hitherto it has been necessary for a person who had reason to believe he had become infected with rabies to proceed to one of the various recognised Pasteur Institutes for the purpose of obtaining treatment. This often entailed a journey of many hundreds of miles, causing considerable delay in the commencement of treatment.

As it is of the greatest importance that antirabic treatment, when required should be commenced as soon as possible after infection, Semple sought for some method by which the vaccine could be prepared in a convenient and portable form whilst retaining its properties unimpaired for a considerable time.

By a careful series of experiments he proved that animals can be as highly immunised with a dead virus as with a living one. It followed, therefore, that if a vaccine could be prepared from this dead virus, it would be both safe and efficient.

The ordinary method of killing vaccines by carefully regulated heat was found to be impracticable for the preparation of an antirabic vaccine.

Recourse was then had to carbolic acid as a means of killing the virus, and after a series of trials with various dilutions of the acid and at varying temperatures, it was found that 24 hours at a temperature of 37° C. was sufficient to kill the virus in 8 per cent. dilutions when 1 per cent. carbolic acid had been added.

After 24 hours the killed vaccine was diluted with an equal volume of sterile physiological saline solution. This gave a 4 per cent. dilution of virus in 0·5 per cent. carbolic acid physiological saline solution: a dilution eminently suitable for treatment purposes.

This vaccine, so prepared, retains its immunising properties unimpaired and will keep for months if stored in a moderately cool place and away from the light. It possesses the great advantage that it can be sent from the place where it is prepared to any part of the country. Medical men, therefore, having a stock of this vaccine at hand would be in a position to commence treatment of a patient infected with rabies at once. Much valuable time would thus be saved, the patient given a better chance of recovery, and without having to undergo the ordeal perhaps of a long and expensive journey to the nearest Pasteur Institute.

H. B. N.

PYOCYANEUS INFECTION IN DOGS AND ITS SIMILARITY TO RABIES.

[Veterinary Record, July 1911.—Major HARVEY, I.M.S., Captain CARTER, I.M.S., and H. W. ACTON, I.M.S.]

AN important paper on this subject appears in the above-named publication. The authors had the opportunity of studying several cases of dogs which clinically showed most of the symptoms of rabies and yet which, in their opinion, did not seem to be true cases of that disease.

In all they studied five cases. No evidence of rabies was found post-mortem in any of the animals. No Negri bodies were made out in the hippocampus major of any of the dogs. Inoculations of brain-substance from the affected animals were made into rabbits or guinea-pigs, and in every case the inoculated animals died with symptoms (wasting, paresis, &c.) similar to those observed in the original animal. No signs of rabies were found post-mortem in any of the experimental animals, but cultures made from the brain, heart, blood, or liver showed, after incubation, typical growths of *Bacillus pyocyaneus*.

In the case of one of the dogs cultures from the heart-blood direct, without resort to subinoculation into a second animal, gave a marked *pyocyaneus* culture in some 48 hours.

It seems evident, therefore, from these investigations that dogs may be the subjects of a *pyocyaneus* infection, and that such infection may, both as regards symptoms produced and tests applied, lead to confusion with rabies.

H. B. N.

THE PATHOGENICITY OF *BACILLUS PYOCYANEUS* IN BRITISH GUIANA.

[E. P. MINETT and W. J. DUNCAN.—Journal of Tropical Medicine, no. 11, vol. xiv.]

THESE investigators point out that the *Bacillus pyocyaneus* is an organism of far more frequent occurrence in British Guiana than in England. In England it is undoubtedly rare to find a case of human infection with this organism, and on studying the reports from abroad the occurrence of this organism is only occasionally reported, chiefly in association with diarrhœa or dysenteric conditions. Thus Williams and Cameron ('Journal of Pathology and Bacteriology,' 1896) record it as associated with diarrhœa, whilst Calmette in Tonkin and Lartigan ('Journal of Experimental Medicine,' iii. no. 6) in New York State have both found this organism in cases presenting true dysenteric symptoms.

Minett and Duncan give particulars of 23 cases in which *B. pyocyaneus* was present on post-mortem examination, usually associated with other organisms. In three of these cases—namely, cæcal ulceration with liver abscess, spreading ulceration of legs, and acute filariasis with suppurating inguinal glands—*B. pyocyaneus* only was found.

Of the full 23 cases recorded in which the *B. pyocyaneus* was found, one is struck with the fact that six of these are cases of acute filariasis, whilst six of the other cases showed intestinal ulceration.

The association of the *B. pyocyaneus* with acute filariasis seems to be frequent in British Guiana, and seeing that none of the cases of this disease recorded by the authorities showed any ulceration through the skin or any evidence of external wounds or abrasions through which the organism could gain entrance, it appears certain that this bacillus must have a great aptitude for migrating about the body until it finds a suitable nidus where it can manifest its baneful properties. The authors give a very complete and detailed list of the cultural and staining reactions of the various strains of *B. pyocyaneus* isolated by them, and their results show that the organism is one which would well repay investigation as to its pathogenicity in other parts of the Tropics. H. B. N.

THE TOXIN OF *BACILLUS SUBTILIS* AND ITS RELATION TO TRYPANOSOMES.

[LEVADITT and TWORT.—Comptes Rendus de la Société de Biologie, no. 15, May 1911.]

THE authors give the results of their investigations into the action of certain bacillary organisms on Trypanosomes.

Various organisms were used, including *Bacillus subtilis*, *Bacillus coli*, *Bacillus prodigiosus*, *Bacillus mesentericus*, and *Bacillus pyocyaneus*. Of these *Bacillus subtilis* alone was found to have any destructive effect.

Working with *Trypanosoma brucei* and broth cultures of *Bacillus subtilis*, it was found that, *in vitro*, when 15 drops of culture of *Bacillus subtilis* were mixed with 2 drops of infected blood at 37° C., complete destruction of the trypanosomes took place in 25 minutes. The question arose whether the destruction of the trypanosomes was due to the direct action of the microbes themselves or to some soluble toxin elaborated by them. It was found that on separating, by filtration, the organisms from the fluid in which they were contained, the filtrate still possessed the trypanolytic action, and, further, that the microbes when isolated and washed were incapable of producing any trypanolysis. The toxin elaborated by the organisms was thus shown to be the active agent in the destruction of the trypanosomes.

The action of this toxin was found to be fairly intense, as it effected the destruction of the trypanosomes at 37° C. in two hours, even when the dilution was as high as 1 in 1000. The amount of toxin present in the broth was found to vary with the age of the culture, the maximum being reached on the second or third day, though cultures 10 days old were still toxic. The toxin is destroyed by exposure to heat at 80° C. It was found to have an equally destructive effect on *Sp. duttoni* and on cultures of the Leishman-Donovan bodies.

Experiments were then conducted to ascertain if this toxin given in large doses to animals infected with Nagana would influence the course of the infection. It was found, however, that this trypanotoxin did not destroy the parasites circulating in the blood, nor did it seem to appreciably hinder their multiplication. Further investigation showed that the toxin rapidly became fixed to certain cells of the organism, especially to the cells of the testes in the rat and to those of the brain, spleen, and kidney in the guinea-pig. The inactivity of the toxin, therefore, in the living body is due to the fact that the poison is fixed by the living cells before it has time to attack the trypanosomes.

Further experiments were then carried out to see if a strain of trypanosomes could be produced which should be toxin-resistant. Varying quantities of toxin were added to the same volume of infected blood and the mixtures incubated at 37° C. The flagellates were examined $\frac{1}{2}$ to 2 hours later. In those tubes which contained sufficient toxin, the parasites, so far as microscopic examination showed, were completely destroyed. The contents of these tubes were then inoculated intraperitoneally into mice. In spite of the apparent absence of living trypanosomes in the inoculation material, several of the mice became infected after about five days.

The sensitiveness of the trypanosomes contained in them was then tested *in vitro*, and these were found to have become resistant to the *subtilis* toxin.

Attempts were further made to ascertain if this resistance could be exalted by several alternations of treatment *in vitro* and passages through mice, but no increase of resistance could be obtained even after six such alternations, nor did repeated passages of the toxin-resistant trypanosomes through mice affect their resistant properties in the least. H. B. N.

VI.—CLINICAL MEDICINE.

TREATMENT OF LEPROSY IN BRITISH GUIANA.

By K. S. WISE, M.B., B.S., B.Sc., D.P.H.,
GOVERNMENT BACTERIOLOGIST, BRITISH GUIANA.

THE cure or alleviation of Leprosy has always been a serious concern of the Medical profession ever since the most ancient times, at which period the disease was regarded as hopeless and incurable.

Vegetable resources have produced many attempts and many countries have been ransacked for the necessary drugs.

In India oils have been prepared and used from the following plants:—*Albizzia libbek*, *Gynometra ramiflora*, *Dipterocarpus turbinatus*, *Gynocardia odorata*, *Hydrocarpus wightoni*, *Hydrocarpus venerata*, *Pongonii glabra*, *Psoralla corylifolia*, *Sunocarpus anacardium*, *Arachis hypogea*. Again, from India comes the powder Madar, from the root of *Asclepias gigantea*. In China and Trinidad a Euphorbiaceous plant, producing a powerful poison in the cortex, has been used in the form of a pill known as Hoang-Nan.

In Brazil there has been used another Euphorbiaceous plant called Ussacon, the Brazilian name of the *Hura braziliensis*.

Venezuela and British Guiana have produced the famous cashew-nut oil, from the *Anacardium occidentale*.

The Andaman Isles give us the gurjun-oil, from several species of *Dipterocarpus*, mainly *lævis*, *tuberatus*, and *trinervus*.

Of the simpler organic and inorganic products a whole host of articles have been tried, amongst others may be mentioned carbolic acid, creosote, glacial phosphoric acid, oxalic acid, pyrogallic acid, phosphorus, arsenic in its various preparations, mercury, iodine, iodide of iron, antimony salts of many kinds, potassium chlorate, potassium iodide, sodium salicylate, kreuznach salts, ichthyol, europhen, aristol, hydroxylamin, naphthol, salol, chrysarobin, and methylene blue.

The more modern serum methods of treatment are represented by the administration of tuberculin, of leprolin made by Rost, and of the nastin introduced by Professor Deycke.

Nerve-stretching, electricity, radium, sea-baths, &c. have each played a part in this problem and have been found wanting.

The majority of these treatments have been tested by Dr. Danielsen at the Lungegaard Hospital, Bergen, in the course of over 50 years' experience of this disease, with, unfortunately, very unsatisfactory results.

In British Guiana, where leprosy is common as a disease and where it presents itself frequently to the practising physician, where 430 lepers are interned in the Asylum and where a much larger number remains outside, the subject has claimed, naturally, a large amount of attention.

British Guiana has long been to the front in combating this disease and has been foremost in voting money for investigating methods of treatment likely to relieve or cure leprosy.

An extremely interesting and full historical account of the Leper Asylums of British Guiana, by Neal, appears in the Journal of Tropical Medicine for 1900, in which he traces the development of Asylum treatment from slavery days to the present time, and from which the following is extracted:— During the slavery period (previous to 1836) each plantation provided for its own lepers, who were relegated to the back dams of the estate, and in this rough manner it was hoped to separate the diseased from the healthy; one finds, however, that the archives of 1831 show 431 lepers in Demerara and Essequebo alone.

The first mention of a Government Leper Asylum occurs in 1832. Lepers were removed to a sand-reef at the mouth of the Pomeroon River in the proximity of the Indian Post.

In 1845 an ordinance to establish Asylums for lepers, and to provide for their care, maintenance, and support, was passed.

According to older residents a settlement was made somewhere above Hyde Park, on the Demerara River, probably on the Himaroonic Creek, which to this day is known as Cocobay (local name for lepers) Creek.

Similarly, it appears that Kaow Island in the Rio Massuruni, opposite H.M.'s Penal Settlement, was used as an Asylum for lepers.

Certain it is, however, that, from 1850 onwards, lepers were located in the Public Hospital, Georgetown, in a part known as the Lazaretto, a low one-storey range on the site of the present Manget wards. In time this accommodation became too small and the Alms House received many lepers, and a separate establishment was built in the present Bourda District somewhere about the end of Regent Street.

These people most naturally must have been frequently in evidence in the streets of Georgetown, and it is not surprising to find that the Lazaretto and Bourda Establishments were abolished and the occupants sent to the newly acquired buildings at Mahaica in 1858, 22 miles from Georgetown.

In 1870 another ordinance with reference to lepers passed the Court of Policy, and Kaow Island was rehabilitated, on the recommendation of the Surgeon-General Manget, to enable Dr. Beauperthuy to carry out his experimental treatment for the alleviation and cure of leprosy.

In 1884 Kaow Island was abolished and one Asylum only retained, viz. that at Mahaica.

The Mahaica Settlement, which was bought from the Imperial Government, had been until then in occupation as a military post. The buildings were naturally not pre-eminently suited for their second purpose and appear to have been badly overcrowded. Three hundred lepers were then housed where less than half that number now live.

Dr. Milroy, sent by the Secretary of State for the Colonies to report on Yaws and Leprosy in the West Indies, reports in 1871 very adversely on the suitability of the buildings and surroundings and suggests Bartica Point and Kaow Island as appearing more satisfactory.

In 1877 with the advent of Dr. Hillis a brighter era dawned. His name will long remain connected with the Leper Asylum of British Guiana, not only as the author of that well-known classical work 'Leprosy in British Guiana,' but also for his able administration of the Institution, in which he took so much interest and in which he worked for over 7 years.

In 1882, in consequence of rioting, the females were removed to Gorchum, 5 miles up the Mahaica Creek, and in 1893 returned once more to Mahaica, when 125,000 dollars were spent in improving the site and buildings. Since that time no great changes have been made, though, as the older inhabitants say (some of whom have been there 30 and 40 years), "It was a nigger yard in those days, but now it is a town."

Between the years 1892 and 1897 no less than seven papers on various aspects of leprosy appeared in the British Guiana Medical Annual, showing that as time progressed interest in this disease far from languishing has, on the contrary, increased.

One of the first systematic attempts at curing this disease was made by Dr. L. D. Beauperthuy, of Cumana, Venezuela, who was invited by this Government to carry out his treatment in this country. Every facility was offered him and Kaow Island refitted and placed at his disposal.

He utilised general sanitary conditions—residence in as salubrious a locality as can be procured, diet nutritious but not stimulating, and special attention to ordinary hygienic rules. Internally bichloride of mercury was administered in small quantities. The external applications used were certain liniments, of which one contained balsam of copaiba to cure the general non-specific eruptions so common in leprotic patients; for active removal of the tubercles nitrate of silver and copper and principally the oil of the cashew-nut. This oil is found in the pericarp of *Anacardium occidentale* and is extracted with ether and evaporated to a thick brownish-black oil.

Unfortunately Beauperthuy died twelve months after arriving here and now lies buried at Bartica.

This treatment had fair trial by the several surgeons attached to the Penal Settlement and also at Mahaica; nothing resulted to justify the expectation held out concerning it.

Chaulmoogra-oil (*Gynocardia odorata*) was widely used by Hillis, who writes that for satisfactory results with this oil the disease should be recent, the patient's health good, untainted by scurvy or syphilis, and that the disease be not hereditarily acquired. Further, the tuberculated are more amenable, the mixed cases next, and the non-tuberculated the least amenable of all.

A dose of ten minims in emulsion and milk or sometimes castor-oil was given internally, while externally a liniment of one part chaulmoogra-oil and fifteen of sweet-oil was used.

Carter in his work 'Leprosy and Elephantiasis' reports that under its continued use the skin becomes soft and supple, the discoloration vanishes, the different morbid sensations leave the patient, the mental hebetude passes away, sensibility is completely, partly, or partially restored, the ulcers heal and cicatrise, though prone to break out again. Having, however, advanced favourably to a certain degree most cases became stationary. No cure seems to have been effected.

Gurjun-oil (*Dipterocarpus lævis*) was used also by Hillis at the Mahaica Asylum on the lines laid down by Dougall, who recommended the use of this oil on the strength of his observations in the Andaman Islands.

Mixed with lime-water it is used externally and internally. Hillis states that "given a suitable case, the disease can be arrested by it and that in a few instances there has been no return of the disease for over two years, but it may nevertheless be premature to say a cure has taken place."

For many years after Dr. Hillis left the chief points in treatment were general hygiene and instructing the leper to take care of himself.

Salicylate of soda long enjoyed a reputation for relieving the repeated attacks of leprotic fever. Gurjun- and chaulmoogra oil were still in intermittent use.

In 1897 Kennard attempted the treatment of leprosy by the injection of mercury perchloride as recommended by Radcliffe Crocker. One cubic centimetre of a fluid containing one-fifth of a grain of this salt was injected intramuscularly once weekly.

Twenty-three cases were thus treated, of which twenty-one were of the tuberculated type; photographs were taken; the bacilli in the fluid from the nodules observed.

Symptoms of mercurialism appeared at the fourth month, so the treatment was omitted for a short period.

After six months Kennard reports that the treatment has no effect on the leprotic fever and does not prevent acute exacerbations of the disease. It has no definite effect on the ulcers or disease of the bones. The bacilli were as readily found in the nodules and appeared unchanged.

The next attempt on a large scale to stem the progress of the disease was made in 1908 by the use of nastin introduced by Professor Deycke. This was used by Irving in May 1908 on five or six patients, none of whom showed any improvement whatever.

In the latter part of 1908 Deycke was invited by the Government of British Guiana to make a trial of his nastin treatment in the Asylum at Mahaica.

As previously, in the year 1870, when all facilities were offered Beauperthuy, so also the resources of the Leper Asylum at Mahaica were placed at Deycke's disposal.

In December 1908 Deycke commenced the administration of this treatment at Mahaica and continued personally to administer nastin and to control its action until May 1909, when he left the Colony. Since that time the treatment has been rigidly continued by Irving on the lines laid down by Deycke until June 1910.

During the five months that Deycke was in the Colony he commenced treatment by nastin on 111 lepers, viz., 69 males and 42 females. These were not selected in any way as being suitable, but were accepted just in the order in which the patients offered themselves and included both early and severe cases.

The treatment consisted of the subcutaneous injection of a solution of nastin with benzoyl chloride in olive-oil. Nastin is explained to be a fatty organic substance extracted from the sheath of a streptothrix isolated from a case of leprosy. This streptothrix is not claimed to have any causative relation to the disease.

The quantity given was one cubic centimetre of B_1 strength once weekly for three months, gradually passing to a strength called B_2. The proportion of nastin in these solutions is not stated.

After this first three months' injection two months without treatment elapses, then the treatment is taken up afresh for another two or three months, to be followed by a rest and re-injection.

Unless these precautions are taken Deycke states that reaction (viz. high temperature and severe general ulceration) are likely to occur, and unless the periods are observed patients become "accustomed" to the treatment and the desired effects are not produced.

In 1909 Deycke published a report of the five months' work which he accomplished in British Guiana, claiming that of 103 cases 92·79 per cent. showed retrogression of the leprous process. And also that "there were a number of cases showing varying degrees of improvement which could easily be observed by the naked eyes."

The nastin treatment was continued on the exact lines laid down by Deycke till May 1910 and in many cases till September 1910.

In September 1910 the nastin treatment had been in use for 21 months on 135 patients and was reported on by the Government Bacteriologist. He stated that some cases have undoubtedly got considerably worse during the treatment, the great majority have remained *statu quo ante*, while a few have improved to such a degree that in three cases the results approximated to recovery.

From March 1910 to September 1911, in the Mahaica Leper Asylum about 70 patients have been placed on a treatment of injections of benzoyl chloride in mineral oil, viz., a treatment similar in every respect to the above nastin process, but with the nastin eliminated. Dr. Minett, the Assistant Government Bacteriologist, who is carefully reporting on the above cases, considers that on the whole the results are rather better.

CASES OF TRYPANOSOMIASIS IN ENGLAND, MAINLY AT THE LONDON SCHOOL OF TROPICAL MEDICINE.

BY C. W. DANIELS, M.B., M.R.C.P.

THE treatment and prognosis in this disease are still so far from satisfactory that a series of cases as seen in England may be of interest. A list including ten of these cases was published by Sir Patrick Manson in the 'Annals of Tropical Medicine and Parasitology,' vol. ii. no. 1, March 2, 1908, and in this paper the further progress of the cases there recorded is given as well as some new ones. It is admitted that in all probability there has been a natural selection in the cases, as those running the most acute course probably die before arrival in England. Our cases, therefore, though the symptoms have been marked, are possibly subacute or chronic, but this applies equally to the early cases before the use of atoxyl was known as to the later ones.

For convenience we usually take the cases in the order of their supposed commencement, not necessarily of their diagnosis. I am much indebted to Sir Patrick Manson for permission to use the notes of his cases in hospital, as well as of others who were private patients. These at some time or other were shown to our students or were in our hospital for special examinations.

Case 1. H. K. Gambia.—The case first described by Ford and Dutton on the Gambia, afterwards treated in Liverpool, is included in this list, as it was the demonstration in Liverpool of the characters of the rash and general symptoms, by Dutton, that led to the recognition of other cases.

Case 2. Mrs. M. Congo.—Was first seen by Sir Patrick Manson on account of peculiar circinate rash noted in 1900 and diagnosed by Brodin in February 1903 by blood examination, when trypanosomes were found.

Case 3. Mrs. S. Congo.—Again first diagnosed by rash and symptoms and the diagnosis confirmed by blood examination in October 1902. The disease apparently commenced in August 1901. The patient died of sleeping sickness November 26th, 1903. Arsenic in various forms, methylene blue, large doses of quinine, &c., seemed to have no effect. The use of atoxyl was not then known.

Case 4. J. M. Uganda.—Had a bite and local swelling on the leg in 1901; began to ail June 1902; trypanosomes were found about June 1903 died of sleeping sickness 1906.

Treated with Liq. arsenicalis and other drugs.

Case.	Initials.	Probable date of onset of symptoms.	Trypanosomes found.	Latest reports as to signs of the disease.	General Result.	Duration of disease in years.
1	H. K. (Ford & Dutton's Case).	....	....	....	Death Jan. 1903—Cerebral symptoms.	$1\frac{2}{3}$
2	Mrs. M.	1900–1901	July 1902	None	Good health.	10–11
3	Mrs. S.	1901	Oct. 1902	....	Death Nov. 1903—Sleeping Sickness.	$2\frac{1}{4}$
4	J. M.	June 1902	March 1903	....	Death 1906—Sleeping Sickness.	4
5	Mrs. G.	?	1902	None	Good health.	9 or more
			Treated with Atoxyl.			
6	Z.	?	July 1905	None	Good health.	over 6
7	Mrs. R.	June 1905	Sept. 1905	None	Fair health.	$6\frac{1}{3}$
8	H. C. C. S.	1905 ?	Feb. 1906	None	Good health.	about 6
9	W. R. E.	Sept. 1906	Jan. 1907	....	Death March 1908—Pneumonia.	$1\frac{1}{2}$
10	E. J. G.	Dec. 1907	Jan. 1908	None	Good health.	4 or more
			Treated with Atoxyl and Antimony either by mouth or by injection.			
11	C. G.	Nov. 1906	Jan. 1907	None	Good health.	5
12	C. V.	July 1909	June 1910	None	Good health.	$2\frac{1}{4}$
13	A. P.	July 1907	July 1907	None	Good health.	$4\frac{1}{4}$
14	D. H. L.	Aug. 1908	Aug. 1909	....	Death 1911—Septicæmia.	$2\frac{10}{12}$
15	Dr. McD.	Dec. 1909	Dec. 1909	None	Good health.	$1\frac{3}{4}$
16	W. G.	Sept. 1909	Oct. 1909	Present	Fair health.	$2\frac{1}{12}$

Case 5.—Another case. Mrs. G., has been frequently examined at the school and always with negative results. She was undoubtedly infected since 1902 (Dutton, Todd), but after a 3 months' course of liq. arsenicalis the trypanosomes disappeared and have not been seen since. She was seen a few months ago and appeared to be in good health.

These five cases were before the discovery by Castellani of the trypanosome, in many cases, of sleeping sickness and the further work of Bruce and others, showing the causal relationship. Cases treated 5: recoveries 2: death with cerebral complications 3.

Cases treated with Atoxyl.

Case 6. Z.—Mild case discovered accidentally by Moffat in Uganda, on blood examination in July 1905. The rash and glandular enlargement were present, but no fever, except some attacks of benign tertian malaria. At first he was treated with trypanroth and liq. arsenicalis, but trypanosomes were found from time to time. On February 5th, 1906, treatment with atoxyl was commenced (1 grain twice a week increased gradually to 4 grains twice a week), with a rapid improvement in his general health. Trypanosomes were not found shortly after the treatment commenced. In August 1906 he was, however, still infected, as a monkey injected with his blood died of trypanosomiasis. In 1907 injections into monkey failed. Early this year he was stated to be in perfect health.

Case 7. Mrs. R. Congo.—Probably the disease commenced in June 1905. Dr. Brodin found trypanosomes in September 1905. In October 1905 she arrived in England with a well-marked rash, an inflamed, swollen, and painful patch on her leg, glandular enlargement, emaciation, and marked debility. On November 22nd, 1905, atoxyl was commenced, starting with small doses twice a week, which were increased to 2 grains twice a week. Large doses caused abdominal pain. The symptoms rapidly disappeared, temperature which had been variable became steady and normal, and she gained weight and strength. She returned to the Congo on 8th March, 1907. Since her return she has had fever and of late menorrhagia, but no return of symptoms of trypanosomiasis. Last seen September 20th, 1911; no trypanosomes were found. This was the first case treated with atoxyl, and this treatment was adopted as a result of the success of the treatment of animals by this drug by Breinl and Thomas in Liverpool.

Case 8. H. C. C. S. Portuguese W. Africa.—First had fever in November 1904 and was invalided for this, arriving in England May 1905. His spleen was much enlarged. In England the fever yielded apparently to quinine but tended to recur. Returned to W. Africa in the autumn. For some time he had fair health, but then had fever and dysentery; was again invalided, and returned to England on February 17th, 1906. On February 22nd there was a rapid increase in the size of the spleen, and the blood was examined by Sir Patrick Manson; no malaria parasites were found, but a considerable number of trypanosomes. He also had the circinate rash and one definitely enlarged posterior cervical gland. As in previous cases, treatment was commenced with 1-grain doses of atoxyl twice a week and increased gradually to 2·5 grains—any increase over this amount caused violent abdominal pain. Trypanosomes slowly but steadily diminished in number, but they were occasionally found for some months. During this period he had two smart attacks of orchitis and also severe dental neuralgia. He was very weak, had tremors, and personally, in July, I gave a very unfavourable prognosis. However, he steadily improved, and in July 1907 had been practically free from fever for five months, was heavier than he had ever been, and felt quite well.

Injections of the blood in August into a monkey and rat gave no results. He went to S. America and continued the use of atoxyl for one year longer. He returned to England in apparently perfect health, and has again gone to South America, where two months ago he was reported as being quite well.

Case 9. W. R. E., 46. N. Nigeria.—September 24th, 1906, began to get fever which resisted quinine in full doses. Arrived in England November 29th. On the way home red patches, with bruised appearance in the centres, began to appear on his skin.—Seen by Sir Patrick Manson January 15th, 1907. The trunk was covered with rings and patches of erythema. Painless enlargement of some cervical, inguinal, and axillary glands. Quick pulse. Feeling of lassitude, not of illness. Trypanosomes were found in the blood. The temperature was little raised, usually about 99° F. at night—once in the first fortnight 100°·8.

On January 25th, 1907, atoxyl in 2-grain doses was given every second day and gradually increased to 7 grains. No abdominal symptoms. This was continued till July at his home. After February 12th no trypanosomes were found in the blood. In July, though there were no other symptoms of arsenical poisoning, symptoms of peripheral neuritis, mainly affecting the legs, developed. These subsided when the atoxyl was discontinued and mercury substituted for a time. Atoxyl in smaller doses (2 grains) twice a week was then resumed. Later it was discontinued for a fortnight and trypanosomes were again found and atoxyl at once resumed. In January 1908 he appeared to be quite well, no trypanosomes were found, but in March he died of pneumonia following influenza. In this case it may be doubted how far death was due indirectly to the trypanosomes, but it has to be remembered that pneumonia is one of the common fatal intercurrent diseases in trypanosomiasis in countries where cases are numerous. It is also noteworthy that the trypanosomes reappeared after nine months' treatment which appeared to have been effective.

Case 10. E. J. G., 40. Bathurst. April 21st, 1908.—Sudden onset December 14th, 1907. Fever for a week followed by series of relapses at about a week's interval. Diagnosed by Dr. T. Hood. Left Gambia February 29th. Had acute pains in legs and arms at first. Atoxyl 2 grains, increased to 3 grains every other day, was given and he had mercury by mouth for concomitant syphilis. Temperature, which had been irregular with nightly rise up to 100°, became normal after injections and no trypanosomes found subsequently. Discharged May 5th, 1908. Continued atoxyl nine months and then went to Ceylon for six months, and no trypanosomes were found there in monthly examinations by Dr. Castellani. He then went to Cyprus for six months. After a year's interval without atoxyl he was re-admitted November 9th, 1909. Trypanosomes again present and he had an irregular temperature; symptoms subsided, trypanosomes disappeared under atoxyl which was continued for a year. Towards the end of 1910 went to Para, apparently in good health. Nothing has been heard of him since.

In these cases the main and practically the only effective treatment was atoxyl. The controlling effect of the drug in all cases was marked and in none did an atoxyl resistant condition develop. Relapses did, however, occur, and, as seen in the last two cases, a course of treatment of over nine months was followed by relapse when the treatment was discontinued. The relapses yielded readily to atoxyl. Results: treated 4; recoveries 3; death 1, intercurrent disease—Influenza and Pneumonia.

Our other cases have been treated by antimony, usually with atoxyl as well, since the demonstrations in lower animals of the powerful effect of antimony compounds by Plimmer and Thompson, and the partial failure of the treatment by atoxyl alone, in the cases then under treatment.

Case 11. C. G., 34. Katanga, Upper Congo.—Good health till November 1906, when he had fever not controlled by quinine. On November 9th started to return to England *viâ* Rhodesia and the Cape. He gave up quinine on leaving the Cape, as it seemed to do no good. On arrival in England, January 5th, 1907, was seen by Sir Patrick Manson. Had then the erythematous rash, slight enlargement of superficial cervical glands, was markedly anaemic and sallow. Trypanosomes were numerous. Atoxyl was given in 1½-grain doses, rapidly increased to 7 grains and occasionally 8 grains. In a few days the trypanosomes disappeared and Dr. Todd failed to find any after a prolonged examination on January 11th. There was a general improvement, the rash rapidly faded, the fever and, to a considerable extent, the debility disappeared. On January 13th and 14th there was again fever, but this time due to benign tertian malaria. Quinine, therefore, in addition to the atoxyl was given. The atoxyl was discontinued for three periods: May 26th to June 12th, June 26th to July 9th, and July 16th to July 25th, when perchloride of mercury was substituted. No trypanosomes were found till May 21st, and at that time he was taking 8-grain doses of atoxyl, and these were given every other day till May 26th. From that date to June 12th daily injections of perchloride of mercury commencing with $\frac{1}{14}$th grain to $\frac{1}{7}$th grain, at which it was kept for five days. Atoxyl was then given in 2½-grain doses every other day. Trypanosomes were found a week later, but on doubling the dose to 5 grains they disappeared. Perchloride of mercury was substituted from June 27th to July 6th and then atoxyl resumed. From July 15th to 22nd, hydrarg. c. creta, 1 grain, three times a day was given, when the gums became tender and atoxyl was again given. On August 11th trypanosomes were again found. On August 16th, parafuchsin in addition to atoxyl was given. The dose given at first was 5 grains three times a day, but it was increased rapidly to 20 grains three times a day and continued till October 13th. Trypanosomes were found till August 29th. Ten days after the last dose of parafuchsin, trypanosomes were again found, and on November 1st and 2nd a very large influx of trypanosomes was noted. On November 8th an intramuscular injection (½ grain) of sodii tartrate of antimony was given, and on the 10th, 11th, 12th, and 15th, in doses of ½ grain, 1 grain, 1½ grains, 2 grains, and 2 grains. There was great local pain, but no constitutional disturbance. The seat of injections continued to be painful and became tense and discoloured; one was opened and an ounce of dark sanious fluid escaped and a grey slough was seen which came away in about two weeks. There was no suppuration, but healing was slow. The other swellings took a long time to subside. The pain was so severe as to render morphia necessary. Hypodermic and intramuscular injections in either weak or stronger solutions both caused tissue necrosis. Tartar emetic was therefore given by the mouth, 2 grains, in two or three pints of water, and this was continued for over a year as well as atoxyl in 5-grain doses every third day. Trypanosomes were not found in the blood, and an injection into a monkey on February 7th, 1908, of blood from the median basilic vein was not followed by any infection of that animal. In spite of this a trypanosome was again found, on a subsequent occasion. The patient returned to Katanga, lived an active life there, and was recently in England apparently in perfect health. He is now in Africa and in good health.

Case 12. C. V., 35. Congo.—July 1st, 1909, stung by an unseen insect on the hand. July 14th, fever. From thence had attacks of fever, at first every two or three weeks, but at shorter and shorter intervals down to five days. Had tremors and muscular tenderness. On May 20th, 1910, glands in neck were noticed to be enlarged and later painful. Two crescent-shaped marks were noticed on the body about this time.

June 26th, 1910, Dr. Mordiglia found trypanosomes in the fluid obtained from the glands but not in the blood. Treatment 7½ grains of atoxyl and orpiment by the mouth. October 4th, Dr. Brodin reports no clinical symptoms of trypanosomiasis and that the cerebrospinal fluid was normal. Dr. Brodin gave an intravenous injection, 5 centigrammes tartar emetic, a few injections 7½ grains atoxyl, and quinine 12 grains twice a week. Since July, when the treatment was commenced, there were no rises of temperature to 100°, though after every two to three weeks there were rises up to 99° for a few days.

December 23rd, seen by Sir Patrick Manson. Soamin 3 grains every three days given. There has been no return of trypanosomes. There are pains in neck and headache, but these are now comparatively slight and she appears to be in good health. Rats were injected with her blood March 11th, 1911, with negative results.

Case 13. A. P.—July 12th, 1907, had to take to bed with fever, but had been ailing for 10 days; only been in Africa since June 23rd. Fever was high, 106°–107°, and continued.—Hospital July 18th. Brodin found trypanosomes July 19th, and atoxyl was given and continued ever since till October 22nd, when seen by Sir Patrick Manson. At the end of 10 days the fever ceased, but recurred slightly every 8 days, and more strongly for 3 days every 21 days. Trypanosomes were still found on that date. Atoxyl 3 grains was given every second day. He had intramuscular injections of antimony simultaneously with C. G. in November with same effect.

December 3rd, 1908, had had a good many attacks of fever and trypanosomes were still to be found in his blood; he was then taking arsacetin, 3 grains, every other day, and a lithium salt of antimony 1½ grains daily. Shortly after this he went to New York, where Dr. Cammack, as the case was not progressing satisfactorily, commenced intravenous injections of tartar emetic, and in June 1911 writes that since beginning these intravenous injections, trypanosomes had been absent from the blood, and fever attributable to trypanosomes, for over 18 months. This was a severe case and the effect of injections of atoxyl, alone or combined with antimony by the mouth, unsatisfactory. Tartar emetic in intravenous injections was highly satisfactory.

Case 14. D. H. L., 35. N. Nigeria.—August 1908, had a boil-like swelling on right knee which did not suppurate, and shortly after his first attack of fever. On August 9th, 1909, had a faint rash and slightly enlarged glands in posterior triangle, and for about 6 months had noticed that slight blows on the arm were painful. Trypanosomes were found in his blood. Treatment, 3 grains atoxyl every three days. Trypanosomes soon disappeared, but were found again March 14th, 1910. He was put on atoxyl, 3 grains every second day, and tartar emetic, 2 grains by mouth daily. Had been treated for pyorrhoea alveolaris; occasionally had considerable pain in the jaw. Was subject to epistaxis and had a severe attack on June 18th. As the toothache was unbearable, had four lower incisors extracted on June 18th. Profuse hæmorrhage occurred that day and steady oozing subsequently. It was checked by plugging of sockets. When seen on June 23rd the mouth was in a septic condition, gums sloughing, cardiac bruits had developed, there were petechiæ on the forearms, legs, abdomen, and back. Cardiac murmurs from ulcerative endocarditis were heard, and varied from time to time. Death ensued on June 29th, 1911.

In this case, as in the case of A. E., though death did not occur directly from trypanosomiasis, seeing that hæmorrhage is not uncommon in that disease, it is possible that both the hæmorrhages and the sepsis were favoured by the previous trypanosome infection.

Case 15. Dr. J. C. S. McD. Ashanti.—December 19th, 1909, had fever for 4 days up to 104°, which did not react to quinine. Dr. Kinghorn on December 21st found trypanosomes and an erythema developed; he left West Africa January 21st, 1910. Atoxyl in $7\frac{1}{2}$-grain doses every 10th day from December 21st. Temperature came down after the second dose. On February 7th there were no symptoms and no trypanosomes were found then or at any subsequent examination. Injections of his blood into rats on February 17th, 1910, had a negative result. From February 7th took soamin, 2 grains, every third day and tartar emetic by the mouth daily. He steadily improved in general health and was allowed to return to West Africa December 15th, 1910.

In this case there were no relapses, either whilst taking atoxyl alone or when he was taking the smaller doses and tartar emetic by the mouth. Probably the early date at which treatment was commenced and possibly the larger doses had a bearing on the steady unbroken convalescence.

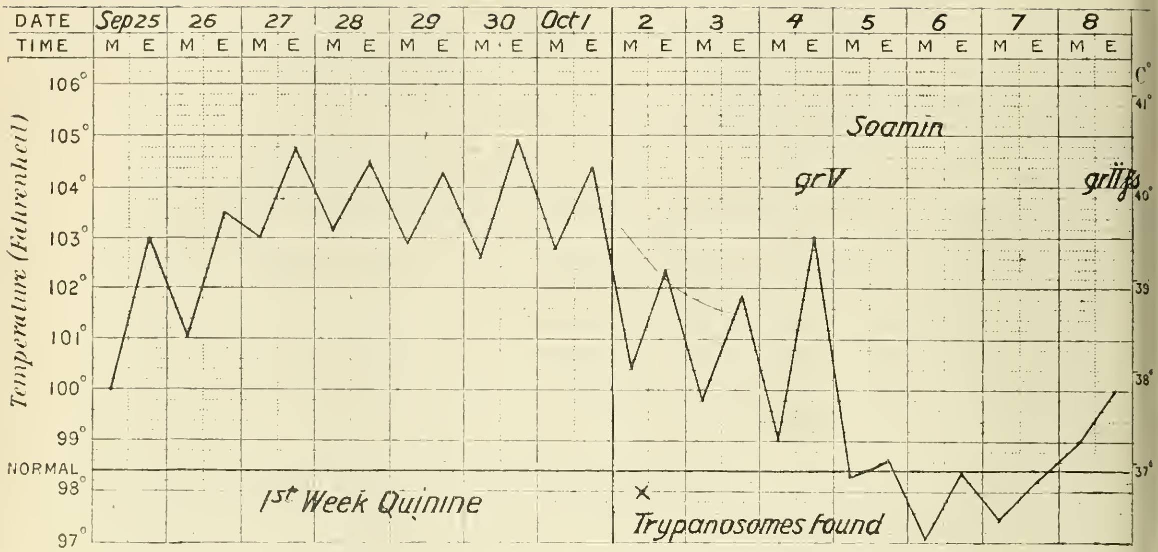
Case 16. W. G. Rhodesia.—Fever commenced on September 25th, 1909, and the temperature chart kept from the beginning somewhat resembled that of typhoid. On October 2nd Dr. Masters found trypanosomes. On October 2nd treatment by soamin in varying doses, $2\frac{1}{2}$–6 grains every other or every third day, and later atoxyl. The temperature though lowered was never normal for 24 hours. During January 1910 had no injections as he was travelling. On February 10th, 10 grains of soamin were given and then $8\frac{1}{2}$ grains alternate days for 8 days, and in smaller doses less regularly; towards evening temperature usually 101° F.

Arrived at the Albert Dock Hospital February 26th, 1910. He was emaciated, weighing 8 st. 6 lbs., bedridden, and to all appearances in a hopeless condition. Atoxyl 3 grains was given every other day, but the evening temperature was usually 101°–102°, and trypanosomes could usually be found easily every day up to March 10th, 1910. He had some unusual symptoms, such as periosteal thickening of the tibia, effusion into both ankles and one knee-joint, and, like case 8, orchitis.

Atoxyl up to this stage appeared to be a complete failure in the doses we had usually given. On March 11th, 1910, an intravenous injection of tartar emetic, $1\frac{1}{2}$ grains, was given, and for 2 days the temperature was normal or subnormal and no trypanosomes could be found for 3 days. Atoxyl was continued in 3-grain doses every third or on alternate days. In spite of this, on the 4th day the temperature rose to over 103° and trypanosomes were more numerous than ever. On March 18th another $1\frac{1}{2}$ grains tartar emetic was injected intravenously. The reaction was very severe, temperature rising to 106°·4, but was subnormal for 3 days, and no trypanosomes were

Chart I.

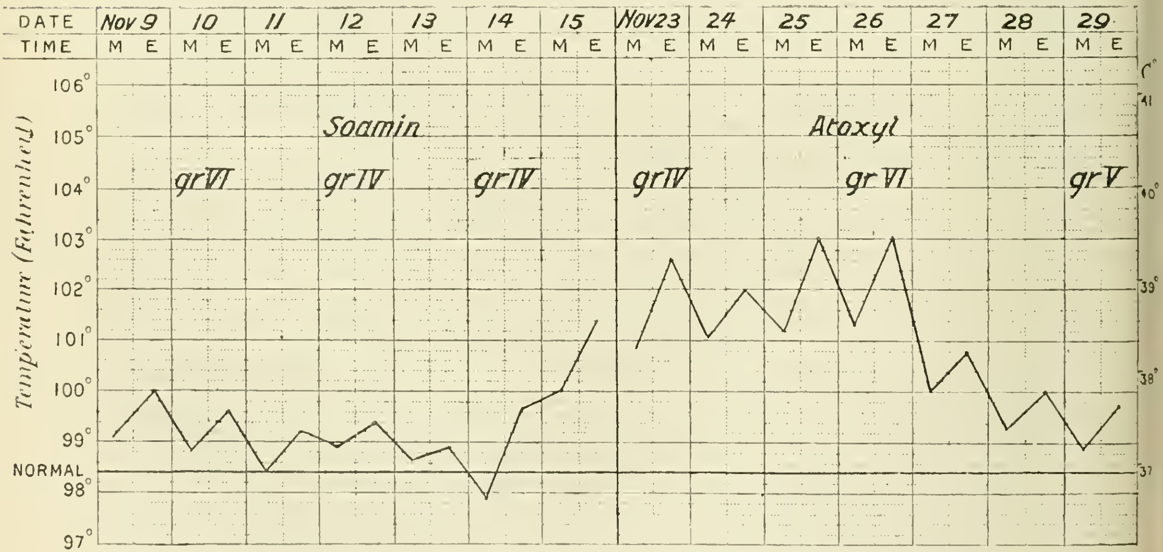
1909.



Onset of disease and marked temporary effect of Soamin in the second week.

Chart II.

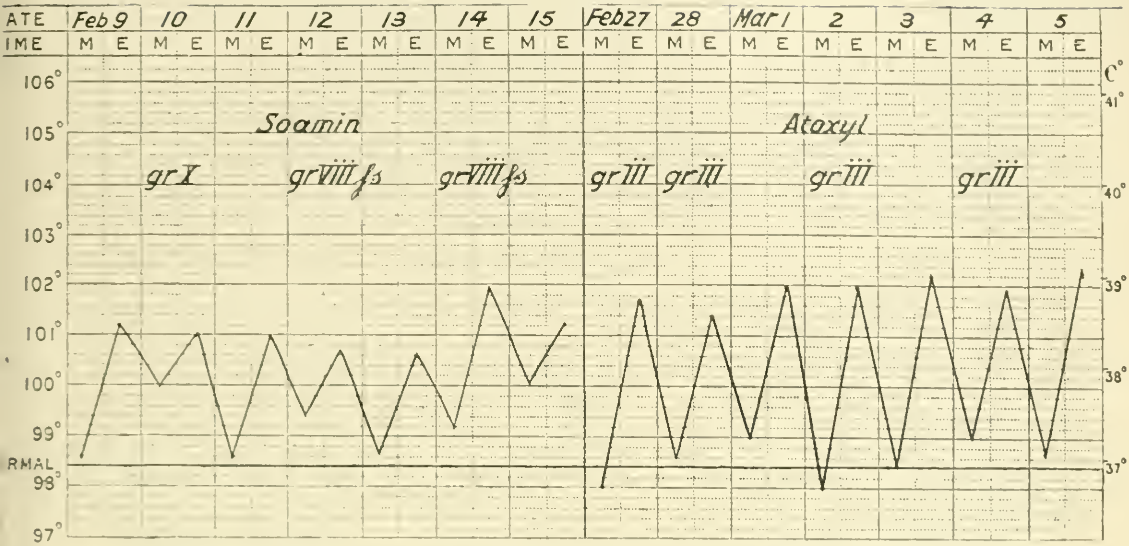
1909.



Third and fifth weeks. Imperfect control of temperature by Soamin and Atoxyl in large doses.

CHART III.

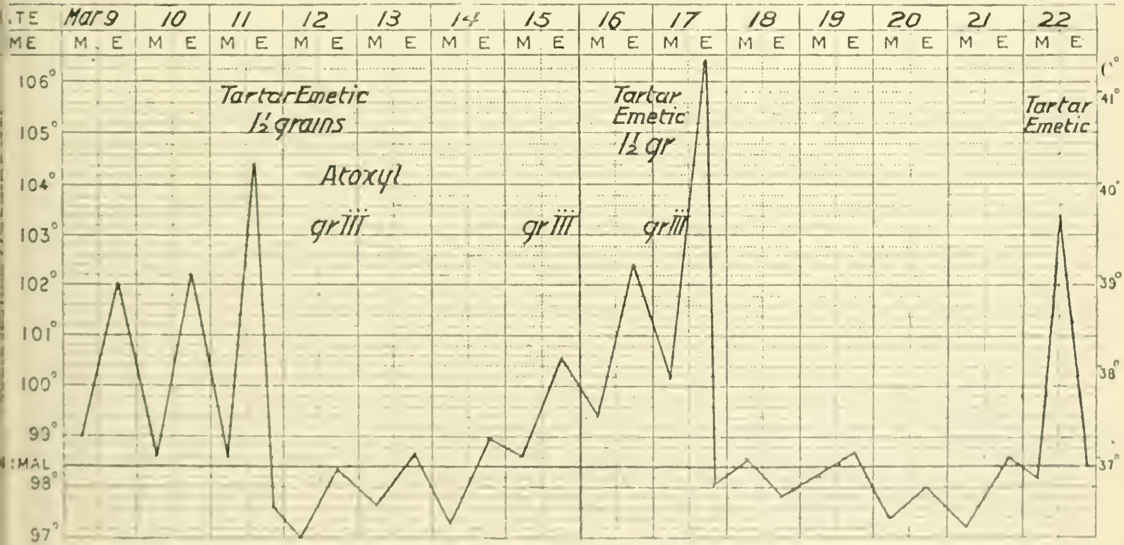
1910.



Two other periods some 3 months later, showing absence of any marked beneficial effect from these drugs. Trypanosomes were usually to be found.

CHART IV.

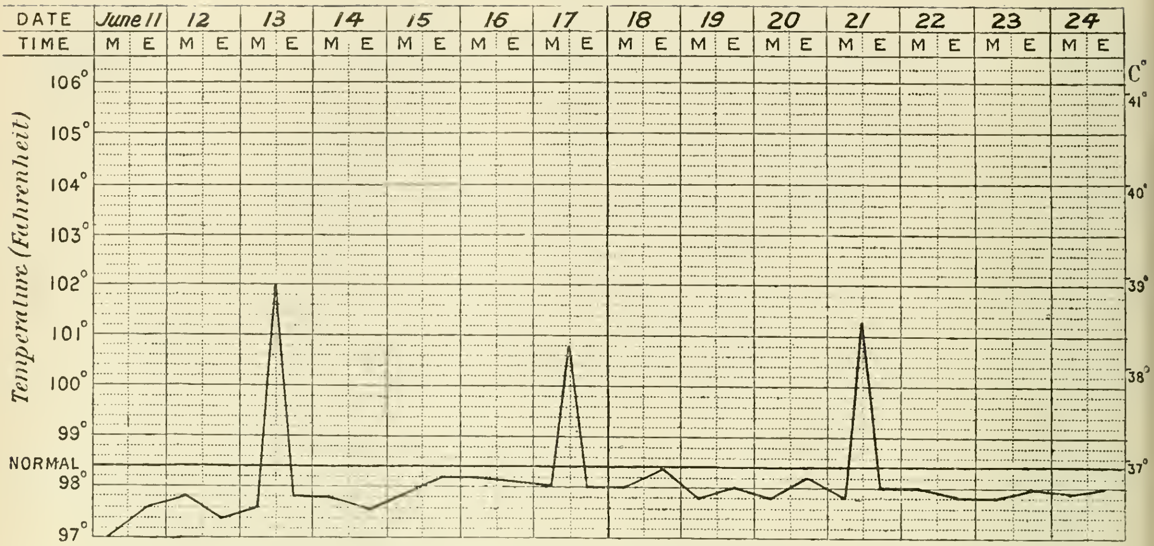
1910.



Marked temporary effect of intravenous injections of Tartar Emetic. The trypanosomes, which before the injection were 3 to 200 leucocytes, were not found for 3 days, but became as numerous as before in 6 days. The severity of the reaction is indicated by the temperature.

Chart V.

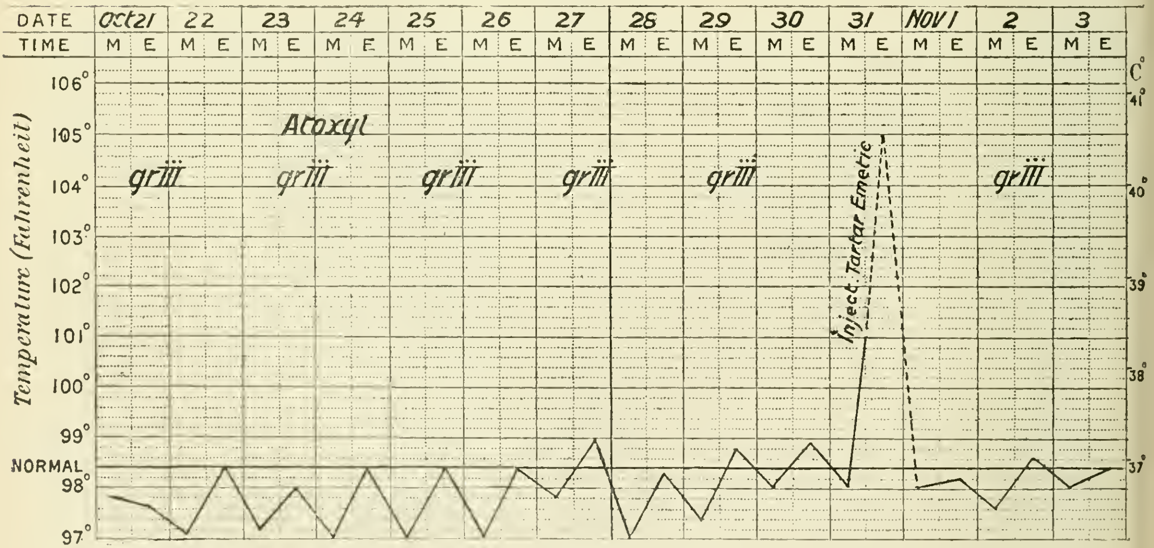
1911.



Tartar Emetic given intravenously every 5th day, but no Atoxyl, for some time seemed to be a success; as the temperature, except for the rises which followed the injections, was normal, and no trypanosomes were found. Ultimately the temperature began to rise on the 5th and trypanosomes were sometimes found on that day.

Chart VI.

1911.



Present condition. Under Atoxyl the temperature remains about normal for 8–12 days and no trypanosomes are found. Then there is a rise of temperature, and, as a rule, trypanosomes can be found. An intravenous injection of Tartar Emetic is followed by a severe reaction, indicated by the dotted line in the temperature chart, and the temperature then falls and trypanosomes cannot be found for another period. The use of Atoxyl now seems to have lengthened the period of well-being from 4 or 5 to 8–12 days.

found till the 4th day, when another injection of tartar emetic was given. Each injection was followed, in addition to the usual pulmonary distress, by a marked swelling of the testicles. In the later injections no such symptoms occurred, but sometimes one or other lymphatic gland would swell or become tender. From this onwards an intravenous injection of tartar emetic, 2 grains, and on the following day an intramuscular injection of atoxyl, 3 grains, were given, and except for the rise of temperature which invariably followed the injection of tartar emetic, the temperature was nearly normal and trypanosomes were not found. On April 14th symptoms of gastritis set in and the treatment was discontinued. At this stage it appeared as if the disease was under control by the combined treatment, but it was not clear that atoxyl had any share in it.

Four days later, April 19th, trypanosomes were again found, and on the 21st there were 3 trypanosomes to 200 leucocytes, so an injection of tartar emetic was given. The same line of treatment as before was then resumed. An intravenous injection of tartar emetic every 4th day, and on the following day an intramuscular injection of atoxyl. The reaction to the injection of tartar emetic was less, and May 26th the rises of temperature were rarely over 100°, and, as a rule, there was then no rigor. On June 7th atoxyl was discontinued, and from May 26th the intravenous injection was given every 5th day. No trypanosomes were found during the whole of this period, and, except for the rise of temperature after the injection, the temperature was normal. The interval was further increased by one day, but the first time this was done, on the 5th day, there was a rise of temperature up to 100°, and on the 6th day when the injection (July 25th) was given the reaction was more severe and the temperature was 103° F. On July 29th a trypanosome was found, the first since April 23rd, or rather more than 3 months, most of it on tartar emetic alone. The interval between the doses was now again reduced to 3 days, but the dose was also reduced from 2 grains of tartar emetic to $1\frac{1}{2}$ or $1\frac{3}{4}$ grains. This was continued till September 1st, and no trypanosomes were found; but as by this time most of the superficial veins had been used, an attempt was made to again increase the interval to 4 days, but on the 5th day, that when the injection was due, two trypanosomes were found. As the shortage of veins was a serious matter and any discontinuance of the treatment was followed by a relapse, as atoxyl alone was a failure and intramuscular injections of tartar emetic were too painful, metallic antimony in Lambkin's medium was tried. Dr. Plimmer kindly supplied the specially prepared antimony, and injections in this medium had in others not been attended by any special reaction. 1 grain of metallic antimony 5 per cent. suspension was injected into the buttocks on September 10th; it was not at first very painful but soon became so, and there was extensive deep tissue necrosis at the site of the injection.

Trypanosomes reappeared on September 19th and rapidly increased in numbers, so that on September 22nd there were 20 to 200 leucocytes: $\frac{1}{2}$ grain of metallic antimony in $2\frac{1}{2}$ per cent. solution in Lambkin's medium was injected intramuscularly, but, in spite of this and of rectal injections of tartar emetic which were retained, trypanosomes were found almost every day. The rectal injections were given daily and increased up to 6 grains. On October 14th $\frac{1}{4}$ grain of Plimmer's antimony suspended in normal saline was given intravenously; it caused less immediate discomfort, but the febrile paroxysm was more prolonged, lasting nearly 24 hours. No trypanosomes were found for 5 days. On October 20th they were again present and

2 grains of arsenophenyl glycin were given, but on the 22nd the trypanosomes were found again. Metallic antimony $\frac{1}{2}$ grain was again given intravenously: the reaction was severe and prolonged, but no trypanosomes were found till November 1st; after that they were present, till on November 3rd an injection of tartar emetic was given, but a trypanosome was found on November 6th. The increasing difficulty in finding suitable veins and the superior and more lasting results obtained in the lower animals had led to the employment of metallic antimony; and so far it was justified, inasmuch as a single dose had a more protracted influence than a single equivalent dose of tartar emetic, but the reaction was so prolonged that in this case the repeated doses obviously required were not considered advisable.

This period may be considered as one in which antimony in various forms alone was employed.

November 6th, Mr. McDonagh, at our request, gave him an intramuscular injection of 606 (now salvarsan) ·6 grammes. The pain caused was severe—there was definite neuritis of both legs. No trypanosomes were found till November 22nd, fifteen days. It is only fair to state that Mr. McDonagh warned us that in a patient who had had so much atoxyl, a condition of arsenic resistance would be present and that the effects of salvarsan would be doubtful. On the return of the trypanosomes tartar emetic injections were again employed November 25th and 29th and again on December 2nd, and this was followed on December 3rd by a $\frac{1}{4}$-grain injection of metallic antimony, on the 5th, 7th, and 9th, 2-grains injection of tartar emetic, and on the 10th, a $\frac{1}{2}$-grain injection of metallic antimony, and on the following day an injection of 2 grains tartar emetic. The idea was that as with more frequent injections the reaction was less that we might obtain the persistent effect of the metallic antimony as well as any transient effect there might be of the more soluble and rapidly acting tartar emetic. No injections were given after the 12th, and no trypanosomes were found till December 22nd, when they were again present and persisted till December 26th, when the same line of treatment was adopted. The intention had been to give the ordinary dose, 2 grains tartar emetic, followed, on the next day, by $\frac{1}{2}$ grain of metallic antimony; and if the reaction was not severe, to follow this with $\frac{3}{4}$ and then with 1 grain. By some mistake, as the reaction with $\frac{1}{2}$ grain was slight (temperature 101°), on the following day the maximum dose $1\frac{1}{2}$ grains of metallic antimony was given. The reaction was severe, there was some collapse and pulmonary trouble. Trypanosomes, however, were not found till January 10th.

It was obvious that this line of treatment was attended with too much risk to the patient, and it had to be abandoned.

On January 12th, 1911, we again commenced with 3 grains of atoxyl every third day, and, somewhat to our surprise, it had a decided effect now on both the temperature and the presence of trypanosomes, which sometimes were not to be found for a week at a time. Salvarsan, intravenously this time, was given again on June 20th; the reaction was not severe, but the trypanosomes returned in a couple of days. We had been warned that with this second injection, after the first had given such poor results, that in all probability the result would be even more unsatisfactory. Since then atoxyl has been given regularly, and when the temperature rises or trypanosomes reappear, as they do in 8 to 12 days, tartar emetic is given intravenously. He appears to be more than holding his own, and has certainly gained in weight (is now 10 st. 7 lb.) and strength, and shows no sign of implication of the nervous system.

This case presents many points of interest:—(1) It exemplifies the greater virulence of the Rhodesian strain of trypanosomes, which was also shown in the inoculated animals: (2) the great resistance to atoxyl, which at first seemed to be absolute, though in the lower animals no marked resistance to the drug was to be noted: (3) that this resistance was in part lost as the case progressed and not increased as might have been anticipated.

Case 17.—C. W. B. In S. Nigeria.—July 1910 had "low fever" with evening rises to 101° and 102°, sometimes 105°, for 19 days. A month later had muscular pains in the back, and at times intense pain at the bottom of the spine for 6 or 7 hours. These muscular pains have persisted ever since, sometimes in one region of the back, sometimes in others. Arrived in England in December, anaemic, and had lost weight—lassitude. In March had an oedematous swelling in right eyelid lasting 3 days, and in April a similar swelling in the left eye. No *Filaria loa* or filarial embryos have been found. Has had several attacks of fever. During the last few months has noticed that the skin got red on slightest friction, but it was not till early in June that there was any definite erythema on any part of the body. He was seen on June 1911 and had a typical trypanosome rash. Enlarged glands in neck and axillae. Kerandel's symptom present. Admitted to hospital July 1st. Temperature showed a diurnal variation, usually of 1½° F., and at night was 99° or more, once 100° F. Tartar emetic was given intravenously in doses of 1½ grains, July 6th, 11th, and 13th—reaction very slight. Highest temperature 102°·2 with the first. After the first injection no trypanosomes were found. Between July 13th and Sept. 16th has had two series of injections of 1½ grains of tartar emetic every 3 days for two weeks, with an interval between them of two weeks. At the end of the interval trypanosomes were again found. On September 16th after the second series there were none to be found, and he reports that there is now no rash and that the glands in the neck are almost normal. He still has the muscular pains. Trypanosomes were again found on October 10th, 24 days after the completion of the last series of injections.

In this case it is doubtful if the muscular pains are not symptoms of trypanosomiasis. On the whole the effect of the antimony was not as good as atoxyl was in the other Nigerian cases, as the temperature did not come down so rapidly to normal and the rash faded very slowly.

General Conclusions.

It is clear from this series of cases that Europeans who can be well fed and carefully looked after have a good chance of at least prolongation of a useful life with the modern methods of treatment, however unsatisfactory the same methods may prove to be in a native population.

They do not give much support to the large early doses of our powerful controlling drugs advocated by some of the authorities, and the fears expressed that we might by repeated small doses produce atoxyl resistant or antimony resistant forms does not seem to have been realised.

That relapses occur even when this treatment is continued is admitted, and if the drug is discontinued too early, as in cases 8 and 9, will occur after prolonged intervals. Some cases are atoxyl resistant to a large extent from the beginning, but even in these, as illustrated by case 16, the resistance to atoxyl is now less definite and more intermittent than it was at first.

Atoxyl combined with antimony, or possibly salvarsan followed by atoxyl and antimony, in fact, the prolonged administration of one or both combinations of antimony and arsenic, appear to be the more promising lines of treatment.

My experience of salvarsan is limited to case 15; but in this it was a failure, as was predicted by the advocates of salvarsan, on account of the previous prolonged treatment with atoxyl.

It will further be noted that as regards the severity of the symptoms, the readiness with which they yield to treatment, and the tendency to relapse there are so great variations that one is not justified in concluding that these differences necessarily indicate different species. In case 16 the infection was acquired in Rhodesia and was not, as far as can be ascertained, acquired from *Glossina palpalis*, but probably from *G. morsitans*; the greater pathogenicity to lower animals was as marked in this as in the Liverpool case, but it is rare to find forms with the morphological characters on which the species *T. rhodesiense* was formed in rats infected with this strain.

Case of Malarial Neuritis.

A. P. B., male, unmarried, aged 37 years, foreman platelayer by occupation, was admitted to the Albert Dock Hospital on the 6th October, 1906, under the care of Sir P. Manson. During the years 1897 and 1898 he had served as a soldier, during the Matabele rising in Rhodesia. From 1897 to 1901 he had served, during the South African War, in the Rhodesian Horse, with Plumer's Column. In 1901 he returned to England for 3 months. From 1901 to 1903, with a period of 4 months' leave to England, he had been employed as a foreman platelayer in Lagos. From 1903 to April 1906 he had remained in England. In April 1906 he went to the Gold Coast.

He had always been moderate in the use of alcohol. When in England his custom was to take about four glasses of beer daily. In Rhodesia, during the war, he had had little alcohol of any kind. In Lagos he used to take "two tots" of whiskey (say a sherry-glassful) daily, and this he took in the evening after 5 P.M. Since April 1906 he has been a total abstainer.

While on the Gold Coast the water he used for drinking was that which had been collected in galvanized iron tanks from the galvanized iron roofs of the Railway Station buildings. He was careful about the collection of this water. He saw himself that the tanks were properly cleaned out. He states that the earlier part of the rainfall was not allowed to run into the tanks. The water was boiled and filtered before use. With regard to the avoidance of malaria, he took no particular precautions in Rhodesia. There he had quinine at irregular intervals. At Lagos he took quinine in 5-grain doses twice a week and used mosquito curtains. On the Gold Coast he took 5 grains of quinine daily and used mosquito curtains.

He never had syphilis. At Lagos he occasionally had fever, but this was never sufficiently severe to prevent him doing his work.

In May 1903, at the Branch Hospital, Albert Dock, he had pneumonia and pleurisy. On the Gold Coast, whilst occasionally "off colour," he had not, so far as he knows, any fever.

In August 1906, whilst at Sekondi, Gold Coast, he was suddenly seized at night with "cramps." He recollects no premonitory symptoms. These "cramps" affected the fronts of both thighs and the fronts and backs of both legs, together with the fingers of both hands. The toes were flexed and he could not extend them except by using his fingers or by pushing his feet against a wall. In the same way he could not extend his fingers; but there was no wrist-drop. He noticed no "knotting" of any of the muscles. The cramps were so painful that he could obtain relief only by continuing to move about on his feet all night. He noticed that when the "cramps" in his legs were bad, those in his hands were somewhat better. When his legs were better, his hands got bad. The "cramps" gradually wore off in about 10 days, but his legs got steadily weaker.

After the attack he continued on duty for about 3 weeks, always in pain, and walking with great difficulty, either by using two sticks or by holding on to articles of furniture. In walking his feet used to droop and come down "with a flop" on the ground. He describes the sensation as similar to that experienced by a person who, going up a flight of steps and imagining that he has one more step to take, essays that step and stumbles because there is none. When the patient sat on the floor, he could raise himself to the upright position only by "climbing up" some article of furniture.

When he touched his lower limbs they seemed "not to belong to him." When he touched other parts of his body the sensation was normal. He felt no numbness of his fingers. There was no sensation of pins and needles. So far as he remembers, pressure on his legs caused no pain; nor were these pains in his hands or legs other than those he attributed to the "cramps."

As he got steadily worse, he reported sick and was invalided to England. He was carried on board the steamer in a hammock. He got somewhat better in the earlier part of the voyage home (as far as the Canaries), then his condition became stationary.

On October 6th he got an attack of fever, and on October 9th was admitted to hospital.

On admission his condition was as follows:—Heart and lungs healthy; liver enlarged; spleen enlarged, felt only on deep palpation; he could walk only if assisted or by holding on to tables or chairs; his feet hung down and dragged along the ground, so much so as to cause him to stumble; he had fairly good power in his hands; pupils reacted to light and accommodation; no loss of tactile sensation; loss of knee-jerk; numbness on front of legs; pain on pressure over nerve-trunks, especially over posterior tibial.

On October 13th it was noted that, for the preceding two days, he had a sore throat and feverish cold. On the night of October 13th–14th he had cramps in his legs. On the night of October 14th–15th he had fever, his temperature rising to 103° F. There was no rigor or sweating. A blood-count was made:—

44	per cent.	polymorphonuclears.
13	,,	large mononuclears.
10	,,	lymphocytes.
2	,,	transitionals.

No parasites were found in the blood.

On October 16th had a temperature of 102°·6 F.

On October 18th subtertian malarial parasites were found in the blood. Intramuscular injections of quinine were ordered. Until after the injections of quinine had been given the patient did not improve; after that the improvement was rapid.

Examined on November 1st the following conditions were found:—

Patient appeared somewhat older than his years; face pale and somewhat muddy coloured, upper and lower extremities wasted : the trunk not so much so. The liver was perhaps a little enlarged : the spleen was not palpable.

The examination, in detail, of the muscular power of the extremities showed :—

Hands.—Movement of thumbs and fingers of both sides normal. Patient able to button a garment with ease with either hand.

Wrists.—Flexion, abduction, adduction, and pronation normal ; neither wrist could be extended to the full extent ; each hand formed, instead of a right angle, only an obtuse angle with the corresponding forearm. Extension was attended, in the case of each limb, with pain along the back of the corresponding forearm. The pain in the right forearm was a little greater than that in the left. Supination was not perfect in either forearm, and in the case of the right limb supination caused a feeling of what the patient described as "straining."

Elbow and Shoulder.—Normal.

Toes.—The toes of the right foot could not be fully extended, but those of the left could ; flexion in both feet was perfect.

Ankles.—The right ankle could not be fully dorsoflexed, the angle the foot makes with the leg remaining somewhat obtuse ; the left ankle could be dorsoflexed more fully than the right—the foot forming an acute angle with the leg. The plantar-extension in both ankles was normal. Eversion of foot was defective on the right side, normal on the left. Inversion was normal on both sides.

Knee.—The flexion on the left side was more perfect than that on the right, the right heel could be brought only to within 3½ inches of the buttock, while the left was brought to 2 inches. The flexion of the right limb was attended with some pain ; there was no pain on the left side ; extension was normal on each side : rotation (after flexion) was better on the left side.

Patient could walk without assistance of any kind ; along a straight line, however, he could only walk (heel and toe) one or two steps.

There was no loss anywhere, in either upper or lower extremity, of tactile or of sensation for pain. There was no tenderness on pressure anywhere except just behind the right fibula in its lower half.

None of the superficial reflexes (epigastric, abdominal, cremasteric, nor plantar) could be elicited on either side of the body.

The tendo-Achilles jerk and knee-jerk were absent on both sides ; rectres clonus could not be obtained on either side; there was no ankle clonus on either side. Wrist-tap and triceps jerk were not obtainable in either upper extremity.

Mental symptoms were absent. He was neither depressed in spirits nor exalted. He conversed freely and, so far as could be made out, his memory, both of recent and of past events, was good.

He was discharged on November 13th, 1906. On discharge he was practically well. His hand-grasp was fairly strong and there was but little defect in his gait.

Note.—Malarial neuritis, though not a very rare condition, is so often complicated by other causes of neuritis, that this case in which treatment by quinine was followed by such rapid results is worthy of record. The notes of the case are by Major J. B. Smith, I.M.S.

Abdominal Diseases.

The latest publication of the Scientific Memoirs by Officers of the Medical and Sanitary Department of the Government of India is devoted to an excellent article on Dysentery and Liver Abscess in Bombay, by Major E. D. W. Greig, M.D., B.Sc., I.M.S., and Captain R. T. Wells, M.A., M.B., I.M.S. In it we find detailed the results of a chemical examination of the cases on admission, with notes of their subsequent progress and post-mortems; also of the examination of stools from dysentery cases, pus from liver-abscesses and blood for Widal reaction: a study of the various strains of the *Bacillus dysenteriæ* and of the amœbæ. The number of cases examined was 397. With respect to the *Bacillus dysenteriæ*, 30·8 per cent. were examples of Shiga's bacillus, the rest of that of Flexner. The main body of their work is devoted to the subject of amœbic dysentery. Their main conclusions embrace the following points, namely: that bacillary dysentery occurs but slightly in Bombay, whilst dysentery associated with amœbæ is more prevalent; that the prevalence of amœbæ shows a marked seasonal variation, following closely the humidity, but having no definite relationship to the temperature curve, and that their greatest prevalence corresponds to the season of greatest prevalence of dysentery: that the special species of amœba was not the *Entamœba histolytica* or *coli*, but that of the variety cultivated by Noc in Cochin China: that these organisms can be cultivated from tap-water and from the stools of a large number of normal animals; that they form cysts both inside and outside the body: and that this cystic shape is important both as regards the treatment and prevention of the disease, whilst dysentery cases should not be considered cured until the stools are found free of the cysts. They find water to be the channel by which the disease is disseminated in enteric areas. With regard to the occurrence of leucocytosis, a high degree is found where there is an extensive ulceration of the colon, and a moderate degree in slight cases. Where there is associated a rise of temperature, an early hepatic invasion by the amœbæ may be indicated, other signs of this occurrence not perhaps being present; these, however, will soon develop and may culminate in abscesses. An important point substantiated is that the administration of ipecacuanha in full doses, as demonstrated by Sir Ronald Ross, will produce a reduction to normal of the temperature and leucocyte curves and prevent the formation of liver-abscess. This is especially important in regard to the treatment of dysentery cases amongst the European troops in India, amongst whom, next to enteric fever, hepatic abscess is the most fatal disease to be dealt with. The work also shows the peculiar difficulties experienced in the interpretation of the results of the cultivation of amœbæ from the fæces: the specific identity of the amœbæ demonstrable in the microscopic examination of the fresh stools, with those appearing in cultures from the same patient, does not appear to be established. A. D.

TETANUS AND INTRAMUSCULAR INJECTIONS OF QUININE.

[Lieut.-Col. Sir D. SEMPLE.—Scientific Memoirs of the Government of India, no. 43.]

THE author gives the results of his investigations on the subject of the occasional occurrence of Tetanus as a sequel to the intramuscular injection of quinine. This accident, which happily now is a rare occurrence, at one time was by no means uncommon, so much so that physicians were inclined to abandon the administration of salts of quinine by means of the hypodermic syringe and to rely entirely on oral administration.

It was formerly considered that the production of tetanus following quinine injection was a result of the introduction of tetanus spores with the injected material, and was really due to using a fouled solution or a dirty syringe or needle.

Semple, however, shows that this is not the whole solution of the problem. He finds that when quinine is injected intramuscularly it has a marked necrotic action on the tissues at the site of injection, these foci of dead tissue serving admirably as anærobic media for the growth of tetanus spores should they by any chance become lodged there.

He was also able to show from experiments on animals that "washed tetanus spores" when injected did not produce tetanus, but remained latent in the tissues for many months. If, subsequently, quinine were injected intramuscularly, tetanus was produced and tetanus bacilli were found at the site of the quinine injection, even if that injection were made into a totally different part of the body to that selected for the original tetanus infection.

He was also able to isolate tetanus bacilli from the intestinal tract of healthy human subjects in four cases out of ten examined.

He comes to the conclusion, therefore, that the production of tetanus as a sequel to the injection may not always be due to faulty technique, but may be caused either by tetanus spores which have been lying latent in the tissues of the body, and are called into action by the tissue-destroying properties of the quinine or by the migration of tetanus bacilli from the intestinal tract to the seat of quinine injection.

A very interesting experiment recorded, as showing the extreme resistance of tetanus spores, was the isolation in a virulent condition of tetanus bacilli from a rusty nib which 18 years before had been dipped into a tetanus culture.

H. B. N.

EPIDEMIC DROPSY.

[Major GREIG, I.M.S.—Scientific Memoirs of the Government of India, no. 45.]

A PRELIMINARY report on the results of investigations into the ætiology and nature of this disease is furnished by Major Greig, I.M.S.

This disease, which seems to have been first described as a distinct clinical entity in Calcutta in 1877, recurs from time to time in epidemic form, and our knowledge of the affection is chiefly due to the studies and writings of MacLeod. An epidemic of the disease occurred in Mauritius in 1880 and is recorded by Sir Francis Lovell, who described the affection as "acute anæmic dropsy."

Major Greig comes to the conclusion that epidemic dropsy is not infectious as was formerly held. He finds no specific cause of the disease in the blood or œdematous fluid, and believes the disease to be a form of beri-beri closely approximating to "ship beri-beri." In the course of his investigations he found that in a mixed community the class chiefly attacked was that whose staple diet was rice, whilst other classes, whose food was more varied, were very rarely attacked.

The chief sufferers were poor Bengalis, whose food consisted practically entirely of rice, whilst other sections of the community, such as the Marwaris (Hindus), whose diet was more varied, escaped entirely, even though the sanitary conditions under which they lived were in no wise superior.

Experiments with various rices were made on pigeons and fowls, and on a diet similar to that consumed by the victims of epidemic dropsy; these birds rapidly showed a loss of weight with characteristic polyneuritis.

Careful chemical and histological examinations of the rice used by the sufferers were carried out by Mr. Hooper, who found that the rice is what is known as "polished rice." In the process of polishing, the outer layers of the grain are more or less removed, and chemical analysis reveals the fact that these outer layers are rich in proteid and phosphorus, so much so that unpolished rice is found to have double the phosphorus content of the polished variety.

Very similar results were obtained by Fraser and Stanton in their work on beri-beri, and it seems to be fairly established that epidemic dropsy must now be classed as one of the diseases due to faulty nutrition and the continual use of a "one-sided" dietary.

H. B. N.

TREATMENT OF A CHRONIC TYPHOID CARRIER.

(Major CLEMENTS and Capt. DAWSON.—Journal of the Royal Army Medical Corps, vol. xvi. no. 4.)

THE discovery made a few years ago that certain individuals after recovering from an attack of enteric fever may for many months, and in some cases even for years, continue, from time to time, to excrete with the urine or fæces, or with both, the specific organism of the disease (*B. typhosus*), in other words become chronic "carriers" of the disease, opened up a new problem in combating enteric fever by preventive measures.

It was found that the organisms were stored up either in the gall-bladder or in little collections in the kidneys, and that from time to time discharges of the bacteria took place from these foci and gained the outer air with the natural excretions.

The problem of dealing with these cases was also rendered the more difficult of solution owing to the fact that no drugs or other means could be discovered which would in any way clear out these bacterial storehouses.

Major Clements and Captain Dawson give a most interesting account of one of these "carriers" and the treatment of the condition with most happy results. The patient, a private soldier, was admitted to hospital on September 18th, 1909, suffering from undoubted enteric fever, but of a very mild type. At the end of the third week his temperature fell to normal, when he was transferred to the convalescent depot. Systematic bacteriological examination of the fæces and urine was commenced on November 19th, 1909, and was continued for practically a year. The examinations were made 5 times a week.

At first the *Bacillus typhosus* was found in the fæces about twice in every ten days, but from February 15th, 1910, it was found almost daily for three months. At no time was the bacillus detected in the urine. The organism was isolated in pure culture from the patient's fæces and a vaccine prepared.

On February 23rd, 1910, an initial dose of the vaccine containing 125 million organisms was administered, and this treatment was continued at suitable intervals and with gradually increasing strengths of dose until May 10th, 1910, six injections in all being given.

No *B. typhosus* were detected in the fæces after June 20th, 1910, in spite of the fact that 110 subsequent examinations were made. An interesting phenomenon noted during the treatment with the vaccine was the occurrence of an acute cholecystitis.

The result obtained is a most encouraging one, and it is to be hoped that treatment on these lines may be equally successful in other cases, and thus provide us with a powerful weapon for eradicating the disease. H. B. N.

REVIEWS.

Mosquito or Man? The Conquest of the Tropical World. By Sir Rubert W. Boyce, M.B., F.R.S. 3rd Edition. 10*s.* 6*d.* (John Murray.)

And by the same Author:

Yellow Fever and its Prevention. A Manual for Medical Students and Practitioners. 10*s.* 6*d.* (John Murray.)

These two books make convincing witness that Tropical Medicine must feel most keenly the loss sustained in the untimely death this year of Sir Rubert Boyce.

The teachings of Sir Patrick Manson and his historic discovery of the mode of transmission from man to man of *Filaria bancrofti* have been the inspiration and guide to like labours of Sir Ronald Ross and those others—captains courageous in the lands of the Tropics—adventurers in the yet wider fields of Science, where lie all the Eldorados. Labours rewarded by great gains to knowledge; priceless boons to humanity. Work which has given a new world to medical science; and to mankind the real conquest of the Tropics. In 'Mosquito or Man,' Sir Rubert Boyce gives a brilliant survey of this brave work, and the great economic profit and saving of human life, following on the practical application in the Tropics of these achievements of Science—the Panama Canal being among the greatest of the first-fruits. The book should be read by everyone in, or in any way connected with, the Tropics, and by all those to whom the Empire is something more than a name. The 3rd Edition has been revised, enlarged, and contains additional illustrations. Of fascinating interest, not the least value of the book lies in the stimulus to the imagination it provides.

Sir Rubert Boyce had had experience of yellow-fever epidemics in New Orleans, Central America, the West Indies, and in 1910 in West Africa. He had taken—to the front as it were—his own expert knowledge of pathology and public hygiene, and a mind informed by careful investigation of the records of yellow fever of more than two centuries; as well as the results of modern researches; the whole applied with energy and enthusiasm —so that he spoke with unquestionable authority on matters concerning this dread disease.

"Yellow Fever and its Prevention" forms an important work. The book is divided into parts dealing respectively with: History and Geographical Distribution of Yellow Fever; Symptomatology and Treatment; Pathology; Epidemiology; Entomology; and Prophylaxis; and contains a wide list of references to the literature, both old and modern, of the disease.

The author draws attention to and quotes from the valuable historic records of yellow fever of Burton, Fergusson, Blair, and Beauperthuy among the older writers and investigators.

With this book, the medical officer in the Tropics is well equipped to meet and tackle the problems of yellow fever; for in it he will find carefully detailed all the methods of dealing with outbreaks of the disease and their future prevention, which have proved so strikingly successful in Havana, Rio, and the Canal Zone, all aptly illustrated by maps and photographs, which increase greatly the value and interest of the book.

The conclusion that yellow fever is endemic in West Africa has excited controversy, chiefly because of the varying interpretations of "endemic." Sir Rubert Boyce's view of the meaning of this term—at all events for West Africa—may perhaps be gauged by the statement, p. 259: "the African native is as saturated with yellow fever as he is with malaria." The evidence for this conclusion is clearly put and is based by analogy on admitted facts in countries where the endemicity of yellow fever has never been in doubt, on the results of wide and painstaking research into the records of yellow fever on the West Coast of Africa—British, French, and German—during the last century; no less than on Sir Rubert Boyce's own experience and observation in the West Indies, the Gulf Ports, and in West Africa in 1910.

This book is indispensable to the medical officer anywhere within the present "Yellow Fever Zone," and those other medical men whose districts are in communication by land or sea with that zone.

If Sir Rubert Boyce took pains to gather all the facts ascertainable bearing on the problems of yellow fever he has also presented them with a literary grace and lucid exposition, lending charm to the reading, and immensely increasing the teaching value of these books. H. M. H.

PERIODICALS IN THE SCHOOL LIBRARY.

Annales de l'Institut Pasteur.
Annales de l'Institut Pasteur de Tunis.
Annali di Medicina navale et coloniale.
Annals of Tropical Medicine and Parasitology.
Archiv für Schiffs und Tropenhygiene.
Archiv für Protistenkunde.
Archives de Médicine et Pharmacie navales.
Archives de Médicine navale.
Archives de Parasitologie.
Archives of Internal Medicine.
Archivos do Real Instituto Bacteriologico Camara Pestana.
Atti della Societa per gli Studi della Malaria.
British Guiana Medical Annual.
British Medical Journal.
Bulletin de la Société Medico-Chirurgicale de l'Indo-Chine.
Bulletin de la Société de Pathologie Exotique.
Bulletin de l'Institut Pasteur.
Bulletin of Entomological Research.
Bulletin of the Bureau of Animal Industry, U.S.A.
Bulletin of the Johns Hopkins Hospital.
Bulletin of the U.S. Public Health and Marine Hospital Service.
Centralblatt für Pathologie und Bakteriologie.
Indian Medical Gazette.
Indian Medical Record.
Journal of Experimental Medicine.
Journal of Hygiene.
Journal of Pathology and Bacteriology.
Journal of the American Medical Association.
Journal of the London School of Tropical Medicine.
Journal of the Royal Army Medical Corps.
Journal of the Royal Microscopical Society.
Journal of Tropical Medicine and Hygiene.
Journal of Tropical Veterinary Science.
Kala-Azar Bulletin.
La Bionutrition.
Memorias do Instituto O. Cruz.
Monthly Report of the Department of Sanitation of the Isthmian Canal Commission.
Paludism.
Parasitology.
Pathologica.
Philippine Journal of Science.
Polyclinic.
Practitioner.
Proceedings of the Royal Society of Medicine (and incorporated Societies).
Quarterly Journal of Medicine.
Revue de Médicine et d'Hygiène tropicales.
Sanidad y Beneficencia, Habana.
Scientific Memoirs by Officers of the Government of India.
Sleeping Sickness Bureau Bulletin.
Transactions of the American Tropical Society.
Transactions of the Epidemiological Society of London.
Transactions of the Society of Tropical Medicine and Hygiene.
Transvaal Medical Journal.
United Empire: and Royal Colonial Institute Proceedings.
United States Naval Medicine Bulletin.
West India Committee Circular.
Yellow Fever Bureau Bulletin.

R. T. LEIPER, *Librarian.*

I.—ENTOMOLOGICAL NOTES.

By A. ALCOCK, C.I.E., F.R.S.

A.—Notes on Medical Entomology.
B.—On some Variations exhibited by two Species of African Anophelines.

A.—Notes on Medical Entomology.

1. *Tsetse-flies.*—Perhaps the most pregnant observations that have been published since the last issue of this Journal are those relating to *Glossina morsitans*. Taute having infected monkeys from sleeping-sickness patients by means of this species, and Kinghorn having transmitted *Trypanosoma rhodiense* in the laboratory through the same species.

Carpenter, in Uganda, and Bequaert, in the Katanga District, have noticed pupæ of *Glossina palpalis* destroyed by small parasitic Hymenoptera of the tribe *Parasitica* (the tribe to which the Chalcid-flies and *Proctotrypidæ* belong); and Simpson, in West Africa, noticed a jumping spider of the family *Attidæ* that appeared to have a predilection for this tsetse-fly. Too much practical importance, however, must not be attached to these observations; for although in dealing with pests of cultivated crops, where the play of natural forces can be controlled and limited, parasites and natural enemies can be utilized with great effect, it can hardly be supposed that *under purely natural conditions* the problem is so simple and easy. Where comprehensive sanitary measures, such as are specified in the 'Bulletin of the Sleeping Sickness Bureau' for January 1909, cannot at once be carried out, more good is likely to accrue by digging up pupæ, by trapping flies, and by experimenting with "tsetsefuges" than by searching for natural enemies and parasites of pupæ.

2. *House-flies.*—The case against the common house-fly as a potential spreader of disease has been made more damaging than ever by the work of Bacot, who has shown that maggots feeding on matter infected with *Bacillus pyocyaneus* may carry on the infection through the pupal stage to the imago stage. The inference therefore is clear that a house-fly, whose life as an adult is of unstained purity, may yet, by reason of defilement in its larval stage, be an infective agent.

A book that should be in the possession of every medical officer is 'The House-fly—Disease Carrier: an Account of its Dangerous Activities and of the Means of Destroying it.' By Dr. L. O. Howard. Published by Frederick A. Stokes & Co., New York.

3. *Classification of* Culicidæ.—Edwards, in the 'Bulletin of Entomological Research' for October 1911, publishes a paper on "The African Species of *Culex* and Allied Genera." Here the African species of Culicines, in which the scales of the head are not flat overlapping squames, are tabulated in 10

genera, the distinguishing characters of the genera being taken from the palpi, the claws, the wing-scales, and the colouring. *Culex fatigans*, Wied., is treated as a variety of *Culex pipiens*, L., a view with which many will agree. The genus *Mansonia* unfortunately disappears, since *Culex titillans*, Walker, which was the type of the genus, was already the established type of Arribalzagia's genus *Tæniorhynchus*. The author gives his reasons for including the species heretofore called *Mansonia* in the genus *Mansonioides*.

4. *Position of the* Corethrinæ.—Annandale, in 'Spolia Zeylanica' for August 1911, describes, under the name *Rancia inepta*, a Corethrine from Ceylon, of which the adult has the veins of the wings covered with *scales*, and the larva has all three segments of the thorax quite distinct. This interesting species not only justifies those who object to the dissociation of the *Corethrinæ* from the *Culicinæ*, but also supports Williston's suggestion that the *Culicidæ* (= *Culicinæ* + *Corethrinæ*) and *Dixidæ* might reasonably be united in one family.

5. *General.*—In 'Entomology for Medical Officers,' published by Gurney & Jackson, London, the author deals systematically with all the groups of insects that affect the human organism, both directly as parasites and carriers, and indirectly by contaminating provisions, &c.

B.—On some Variations exhibited by two Species of African Anophelines.

The fact that the term "species" implies merely a *definition* of the common features of a group of individuals and is an idea rather than an entity, is so often ignored, with the result that "species" are multiplied to confusion, is my justification for drawing attention to the numerous slight deviations from accepted specific standard exhibited by two very common African Anophelines, namely, *Anopheles* (*Myzorhynchus*) *mauritianus* of Grandpré and *Anopheles* (*Nyssorhynchus*) *pharoensis* of Theobald. These deviations illustrate the risks of defining a "new species" from a single individual.

It must be premised that the individual specimens examined are all uncommonly well preserved, all come from one geographical area, and were handled by a keen and experienced collector, Mr. W. van Eeden.

a. Anopheles (Myzorhynchus) mauritianus, Grandpré.

This species and *Anopheles* (*Myzorhynchus*) *paludis*, Theobald, are distinguished from all other species of the subgenus *Myzorhynchus* in having the distal segments of the tarsus of the hind legs snow-white: in *paludis* the 3rd, 4th, and 5th tarsal segments are entirely snow-white, but in *mauritianus* there is a broad black band at the base of the 3rd tarsal segment.

The established specific characters of *Anopheles* (*Myzorhynchus*) *mauritianus* appear to be the following:—

(*a*) The palpi are black, with 2, 3, or 4 rings of white scales;
(*b*) There are two small white costal spots on the anterior margin of the wing: the larger of these two spots lies near the tip of the wing, the smaller lies a little outside the level of the origin of the 3rd longitudinal vein;

(*c*) Except for a yellowish patch at the tip of the wing the wing-fringe is not spotted;
(*d*) The tips of the 1st, 2nd, and 3rd tarsal segments of the front and middle legs are white:
(*e*) In the tarsus of hind legs the 1st segment is white at base and tip, the 2nd is white at tip, and the greater part of the 3rd and the whole of the 4th and 5th segments are white.

In six beautifully-preserved specimens collected—four of them at the same place and on the same day—by Mr. van Eeden, the following variation is apparent:—

(*a*) *As to the palpi*: (1) there may be 3 or 4 narrow but well-defined white rings, one of which includes the tip of the palp: or (2) there may be 1 or 2 white rings, the distal half of the terminal segment of the palp being white; or (3) the palpi may be black except for a *few* white scales at the tip of the 2nd and 3rd segments and a good many white scales scattered in the distal half of the terminal segment. In any case the proximal segments of the palpi may be either black or finely peppered with white.

(*b*) *As to the wings*: the two small white costal spots of the anterior margin (1) may be quite conspicuous on both wings; or (2) may be quite inconspicuous on both wings; or (3) the smaller—anterior—spot may be indistinct on one wing and absent from the other. The colouring of the wing-fringe shows no variation.

(*c*) *As to the front legs*: there are always three broad white bands—one at the tip of each of the first three tarsal segments; but the last two tarsal segments either may be quite black, or one or both of them may have some white scales at the tip.

(*d*) *As to the middle legs*: there are always three bands—one at the tip of each of the first three segments; but these bands may be either broad and conspicuously white, or narrow and merely pale.

(*e*) *As to the hind legs*: the last two tarsal segments are always snow-white and the proximal extreme of the first segment (metatarsus) is always white; but the amount of white on the third segment varies, from a little more than half to two-thirds.

In the six specimens examined, therefore, the most constant specific features are: (1) the snow-white fourth and fifth tarsal segments of the hind legs; (2) the small white costal spot near the tip of the wing; and (3) the wing-fringe being dark except for a yellowish patch at the tip of the wing.

b. ANOPHELES (NYSSORHYNCHUS) PHAROENSIS, Theobald.

("*Cellia*" *pharoensis*, Theobald.)

The following are the specific characters that are regarded as important:—

(*a*) The palpi are thickly covered with brown scales, among which, on the dorsal surface, white scales are scattered, and the tips of the 2nd–4th segments are white-scaled;
(*b*) There are two black spots on the scutum, which is otherwise brown with "cinereous reflections";

(*c*) On the front edge of the wing there are three principal large dark spots, of which the middle one is the largest, and the wing-fringe is dark with a small yellow spot at the end of every vein ;

(*d*) The legs are much mottled and irregularly banded dark brown and yellow, the tarsi are broadly banded with white distally, and the fifth tarsal segment of the hind legs is all white.

Eight well-preserved specimens, four of which were collected on one day in one place, show much variation in the markings of the palpi and the colouring of the scutum.

(*a*) *As to the palpi.* In one specimen they are brown mottled with white, and are not at all ringed; in another they are mottled with white, and two very faint whitish rings can be made out ; in another they are mottled with white, and there is a well-defined white ring at the tip of the third segment; in two others the white scales so much predominate that the palpi appear to be uniformly frosted; in another the brown and white scales fall into irregular and incomplete, narrow, alternating rings ; in another there is the same irregular and incomplete ringed appearance, but the alternating " rings " are broad; in another the white scales greatly predominate, and the brown scales are arranged in a suggestion of 4 or 5 rings.

(*b*) *As to the colouring of the scutum.* In all cases the scutum is covered with broadish light-coloured scales. Beneath the scales the scutum in three specimens is ash-grey with a dark brown median streak and two small very dark brown spots ; in three others it is uniformly dark brown, but the two spots, which are quite black, can be distinguished; in two others it is so uniformly darkened that the spots cannot be distinguished.

(*c*) In the specimens with the very dark scutum the mottling and banding of the legs is much more vivid, the dark markings being almost black.

II.—DEPARTMENT OF PROTOZOOLOGY.

Some Recent Advances in our Knowledge of Leishmaniasis.

By C. M. WENYON.

The most important recent advances in our knowledge of the diseases due to leishmania have been, firstly, the discovery that a form of Kala Azar, which chiefly attacks young children, occurs along the northern and southern shores of the Mediterranean, and that a form of canine leishmaniasis has an almost identical distribution: and secondly, that the disease long known as "Oriental Sore" occurs in South and Central America. That Infantile Kala Azar occurred in Europe was first demonstrated by Pianese in 1905, who found leishmania in children who had died of the diseases long recognised by Cardarelli, Somma, and Fede as the febrile form of infantile splenic anæmia. In the year before (1904) Laveran found leishmania in films made by Cathoire from the spleen of a nine-month old infant which had died of some ill-defined malady in Goulette (Tunis). In 1908, Nicolle and Cassuto described the first case of Infantile Kala Azar in Tunis, and since that time Nicolle and his collaborators have reported in all twenty-five instances of this disease, which has been recorded also from Algeria by Lemaire and from Tripoli by Tahsim Ibrahim. In Europe, following the discovery of Pianese, Gabbi reported, in 1908, the first case in a child from Messina. In the following years other cases were described by Gabbi from Messina, Feletti and his pupils from Catania, and Jemma from Palermo. Cretien found the disease in Malta, Alvarez in Lisbon, and Gabbi showed that the disease known as Ponos, and occurring in the islands of the Greek Archipelago, was none other than the same Infantile Kala Azar. Owing to the work of Christomanos and others, Infantile Kala Azar is known to be widely spread in Greece and its islands. So that this disease, unsuspected a few years ago, is now known to have a wide distribution along the northern coast of Africa, throughout Greece, Italy as far north as Rome and Sicily, Portugal (Lisbon), Malta, Crete, and many of the small islands around Sicily and Greece, and Marzinowsky has reported the disease from Russia.

This disease, though it specially attacks children of two to three years of age, is not limited to them, as cases in adults have been described by Gabbi (ages 18 and 38), by Fulei and Basile in a youth of nineteen in Rome, by Tahsim Ibrahim in a youth in Tripoli, and in several adults by Christomanos in Greece. However, the number of young children from one to four years of age is well over 90 per cent. of the total number of cases so far recorded, and this is one of the chief points in which there is a difference from the Indian Kala Azar, where the proportion of adults attacked is greater.

The symptoms of Infantile Kala Azar are similar to those of the Indian form of the disease. Usually, the first symptom noted is some gastro-intestinal disturbance accompanied, or not, by slight fever. The child recovers from this first attack, but soon shows signs of failing nutrition. It becomes anæmic, loses flesh, suffers from irregular attacks of fever, while the spleen gradually increases in size and may eventually attain enormous proportions. The liver is enlarged, but to a less extent. When the disease is well established there is developed a peculiar pallor of the skin, which Nicolle describes as being so characteristic that one may suspect the disease from this sign alone. The child, if old enough to run about, ceases to do so, and no longer joins in the games of its comrades ; it becomes more and more emaciated, while the abdomen becomes increasingly prominent; the attacks of fever, which conform to no known type and are unaffected by administration of quinine, are more numerous, while the onset of an enterocollitis may threaten the life of the patient. Transitory œdemas about the limbs and face are frequently noted, and punctiform hæmorrhages in the skin are often seen. A condition of hæmophilia with bleeding from the bowel, gums, or nose may develop, and this may be so severe as to cause the death of the child. Ulceration of the intestine as shown by dysenteric symptoms, ulceration about the gums and lips sometimes developing into noma, may contribute to bring about a fatal issue. Death results, as a rule, from the onset of broncho-pneumonia or severe diarrhœa or dysentery. Nicolle has reported several cases where death was due apparently to the sudden onset of a dyspnœa, possibly due to an œdema of the glottis. In other cases it results from severe hæmorrhage from the nose or bowel or the toxic effects of noma. The symptom of fever is apparently never wanting, and its great feature is its irregularity. There occurs febrile periods alternating with periods of apyrexia. During the febrile periods the fever may be intermittent or remittent, and it may suddenly change from one of these types to the other. There may be two or three elevations of temperature in the twenty-four hours. Occasionally the onset of fever is accompanied by rigor and followed by sweating. During one febrile period the type of fever may be quite different from the type it will follow in the next period.

A blood examination shows a reduction in the number of red blood corpuscles, which may fall as low as 1,250,000, while the hæmoglobin reduction is usually in the same proportion. The number of white corpuscles varies very much, for though it is often normal, or above normal, most usually there is slight leucopœnia in which the proportion of mononuclears is increased.

The duration of the disease varies considerably. Sometimes the course is exceedingly rapid, and death may result within forty days of the first noticeable sign. Usually the disease develops more slowly and may last two, three, or four years. The disease is not invariably fatal, as several cases of spontaneous recovery have been reported.

As regards the diagnosis, most observers are agreed that the discovery of the parasite is the only sure means of attaining this end. Nicolle has found the parasites in the blood of the peripheral circulation by a method of centrifugation, but, as a rule, this method gives a positive result in only a small percentage of cases. Spleen puncture is the method most usually employed, though Pianese has removed marrow from the head of the tibia. Nicolle, who has had a great experience in this disease in Tunis, remarks that spleen puncture may be performed with ordinary precautions without danger, and he has repeated the puncture several times in some of his cases.

Within the body the parasites are to be found in greatest number in the spleen, bone-marrow, and liver. They may also be found in the kidneys, lungs, lymphatic glands, intestines, and exceptionally in the muscles. They occur in large mononuclear cells or macrophages, which are possibly derived from the endothelium of the blood capillaries.

As regards its structure, the leishmania of Infantile Kala Azar does not differ in any way from that of the Indian form of the disease. It is culturable in citrated blood, the medium of Novy and MacNeal, the modified blood agar of Nicolle, or the sterilised blood agar of Mathis. In these cultures the parasite is found to develop into a flagellated organism of the Herpetomonas type.

A point of great interest in connection with the Infantile Kala Azar is its almost constant association with a form of leishmaniasis in dogs. In practically all the great centres of Infantile Kala Azar—viz., Tunis, Algeria, Lisbon, Malta, Sicily, Rome, Greece—the ordinary domestic dog has been found to be liable to this disease. This fact was first demonstrated by Nicolle in Tunis, and he propounded the theory of the canine origin of human Kala Azar and suggested that the dog, in which the disease may be exceedingly mild or latent, acts as a reservoir for the virus, which is transmitted to man by some biting arthropod. Basile, working in Sicily and Italy, claims to have proved that it is by means of the dog flea (*Ctenocephalus canis*) and the domestic flea (*Pulex irritans*) that the disease is transmitted amongst dogs and from them to man. Basile's experiments are of four kinds. First, he introduced uninfected dogs into houses in which Kala Azar cases occurred, and states that the dogs contracted the disease: secondly, healthy dogs were made to live in close association with an infected dog on which were many fleas: these dogs also became infected: thirdly, fleas taken from infected houses in Bordonaro, in Sicily, were transported to Rome and there placed upon uninfected and isolated dogs, which very soon became infected: and fourthly, the dog flea (*Ctenocephalus canis*) fed upon spleen-juice from an infected dog is found to become infected with cultural forms of leishmania. The gut-contents of the fleas are infective to healthy dogs. Basile claims to have taken every precaution to exclude errors from his experiments and to have thus established that the flea is the transmitting agent of human and canine Kala Azar. The experiments of Basile have, however, been severely criticised by Gabbi, who has failed to confirm these results. On the other hand, owing to the results of Franchini, who has infected Anophelines by feeding them on cultures of leishmania, Gabbi is inclined to incriminate the mosquito as being the bearer of this disease. Basile in Sicily, Sangiorgi in Turin, where canine leishmaniasis has recently been discovered, and Alvarez in Lisbon, have found in fleas taken from infected dogs flagellates which they claim to be developmental forms of leishmania. These observers declare that fleas which have not had an opportunity of sucking infected blood are found to be free from these flagellates. Though in experiments of this nature one has to be exceedingly cautious in judging of the significance of intestinal flagellates of insects, which, as every one knows, may be simply harmless parasites peculiar to the insect in question, still there is no *à priori* reason why leishmania should not develop in the intestine of the flea or that the flea should not be able to transmit the disease. However, these interesting experiments need further confirmation. It may be mentioned that Sangiorgi, in Turin, noted that a dog which had been living in close contact with an infected dog contracted the disease, and that Marshall, in the Sudan, has had a similar experience, though in this case with an uninfected monkey which was caged with others suffering from the disease.

Experimental inoculation of Infantile Kala Azar was first accomplished by Nicolle in Tunis, who found that both dogs and monkeys could be infected by intraperitoneal or intrahepatic injection of material obtained from cases of the disease. The disease in monkeys resembles that in children more closely than does the disease in dogs, in which it may be exceedingly mild. As a rule, other animals are not infected, though a slight and transitory infection may take place in guinea-pigs and rats. The Infantile Kala Azar of Sicily and Italy is equally inoculable into dogs and monkeys, and in one case Franchini produced a fatal infection in a guinea-pig; and Volpino inoculated a rabbit in the cornea with the result that there was produced a local infection resembling the lesion of Oriental Sore. In Lisbon, Alvarez has also infected dogs. The ease with which the Mediterranean disease is inoculable into dogs has been raised as an argument against the identity of this disease and that of India. Though inoculation of dogs in India has been attempted, the number of experiments is limited and they were not exhaustive. In this connection Nicolle remarks that when two such dissimilar diseases as Infantile Kala Azar and Oriental Sore are inoculable to dogs one would be very much surprised to find that Indian Kala Azar, so similar to the Infantile disease, could not thus be reproduced.

Kala Azar, first shown to occur in the Sudan by Neave, has been found, through the investigations of Bousfield, Marshall, and Thomson, to be widely distributed along the Blue Nile and occurring in the Sennar and Kassala Provinces. In Sennar the disease attacks children with an average age of twelve years, while in Kassala adults are more often affected. A feature of the investigations of the disease in Sennar is the discovery of leishmania in the peripheral blood of 86·6 per cent. of the cases. Marshall has been able to reproduce the disease in monkeys, but so far has not found spontaneous infection of dogs nor has he infected dogs by inoculation.

Though of recent years there has been much advance in our knowledge of the distribution of Kala Azar and the discovery of the disease in dogs which opens up the possibility of their acting as reservoir hosts, little progress has been made in the way of treatment, though most of the important remedies which have been tried for sleeping sickness have here been employed. It is true, however, that certain cases may recover spontaneously; and in this fact Nicolle finds encouragement, since, in his opinion, it shows that the human organism is naturally resistant to a certain extent, and that it needs only the assistance of some therapeutic agent to overcome the parasite. What this agent is still remains to be discovered.

Oriental Sore, up till a few years ago, was thought to be limited to the East, as its name implies. It had been known for years in India, Persia, Asiatic Turkey, Russia, and Northern Africa. It has recently been described from Italy, Sicily, and Greece, and several cases have been recorded from New Caledonia. In 1909 Lindenberg and Carini and Paranhos demonstrated leishmania in an ulcer in Brazil. In the same year Tonin, Heckenroth, and Natan-Larrier established the fact that the disease known as Pian-Bois in French Guiana was a form of dermal leishmaniasis. The disease was then described from Manaos on the Amazon and by Darling in Panama.

Carini and Splendore have lately called attention to the fact that the leishmania may not only infect the skin but that the disease may occur in the mouth and nose, producing therein extensive ulceration and tissue destruction. In most of these cases there is some primary skin-infection, the disease spreading thence to the buccal and nasal cavities. In some cases, however, it is stated that the primary lesion may be in the mouth. This form of leishmaniasis occurs side by side with a form of mycotic infection of the mouth and nose, and the two diseases are readily confused. Diagnosis is made by the discovery of leishmania in the exudation from the ulcers or vegetations in the mouth.

Flu has recently described the disease which occurs in the three Guianas under the names of Forest-Yaws, Boch-Yaws, or Pian-Bois. This is a leishmaniasis, and effects chiefly the skin on exposed parts of the body. It corresponds in many ways with the Oriental Sore of the East, and the author is convinced that the disease is the result of the bite of a tick, as only those who have been working in the bush where ticks abound become infected. The author, however, brings forward no experimental proof of his contention, though the earliest lesion of the disease resembles very closely the wound inflicted by the tick.

Experimental work on Oriental Sore has been undertaken by Nicolle and Manceaux. These authorities have discovered that Oriental Sore may be reproduced in dogs and monkeys by inoculating the skin about the ears, eyes, and nose. The resulting lesion resembles the typical sore in man, but endures a shorter time. They bring forward a certain amount of evidence to show that animals which have recovered from an infection of Kala Azar are immune against the organism of Oriental Sore.

Culture of the parasite of Oriental Sore (*Leishmania tropica*) was first obtained by Nicolle on blood agar. These cultures remain infective and give rise to sores when inoculated into skin of man, monkeys, and dogs. Intrahepatic and intraperitoneal injection of large quantities of culture of *Leishmania tropica* into dogs does not bring about any general infection, though Novy has shown that similar inoculation of dogs with cultures of the leishmania of Infantile Kala Azar gives rise to the disease.

As regards the transmission of Oriental Sore no conclusive results have been obtained. Most observers agree that the house-fly must play some part in this transmission: and Row has found that the parasite may remain infective for monkeys for three hours after they have been taken up by the fly, and suggests that the parasites may in this way be passed in the faeces on to some abrasion of the skin and give rise to the disease. Working on the Bagdad Sore the present writer found that bed-bugs and *Stegomyia fasciata* would feed from the sore and that in so doing parasites were taken up. In the guts of these arthropods the parasites underwent a development into flagellate forms as they do in cultures. Attempts at transmitting the disease to himself by the bites of *Stegomyia fasciata* previously fed upon the sore were unsuccessful, as were also a limited number of similar experiments conducted later in Aleppo with *Phlebotomus*.

The exact relation of Oriental Sore to Kala Azar is still doubtful. Some authorities maintain that the one disease is in reality a mild form of the other produced by an altered or attenuated virus, while others claim that the organisms are quite distinct though resembling one another morphologically. Still more difficult is the question of the relation of the Indian to the Mediterranean Kala Azar. In symptomatology there is a great similarity in the two diseases. The Indian disease is generally more rapidly fatal than the Mediterranean form, though, as already mentioned, this may be fatal in forty days from the onset of the disease. A further difference is the fact that in the West young children of from one to four years of age are most usually attacked, while in India adults are most commonly affected. This discrepancy may be due to the difference in the conditions of life in the two parts of the world and the possible difficulty of obtaining information as regards young children in India. The fact that dogs are so readily infected with the

Infantile Kala Azar while they are apparently refractory to the Indian disease has been frequently raised as an argument against the identity of the two diseases. The Indian experiments, however, need repeating and extending, for it may be that the pariah dog of India is immune, as is suggested by the failure to find any case of spontaneous leishmaniasis in Indian dogs, though over one thousand were examined by Donovan. It will be necessary to inoculate dogs with the Indian parasite under the same conditions as those which are successful in the Mediterranean disease. Till this work is done it is impossible to decide finally whether the two diseases are identical or not. Cases of Kala Azar have been reported from Russia, and Sluka has described a case from Tashkent in Turkestan. It is probable that with further observation the disease will be found to have a wider and extending distribution, so that the idea that any extensive barriers separate the endemic centres of Europe, India, China, and the Sudan will have to be given up.

NOTE ON THE OCCURRENCE OF HERPETOMONAS IN THE PHLEBOTOMUS OF ALEPPO.

By C. M. WENYON.

DURING the course of investigations on the Oriental Sore of Aleppo in the summer of last year numbers of sand-flies (*Phlebotomus*) were dissected. It was found that in a small percentage (about six per cent.) of those dissected there occurred a flagellate of the genus *Herpetomonas*. This flagellate occurred as small rounded or bean-shaped bodies resembling leishmania, and also as elongated flagellate forms like the cultural forms of leishmania. All intermediate stages of development between the small non-flagellate bodies and the fully developed flagellates occurred. The latter had a body varying in length from 15 μ to 25 μ, and a flagellum which was either longer or shorter than the body. The nucleus was centrally placed and the kinetonucleus as a deeply staining granule near the anterior end of the body. The flagellum took origin in an achromatic granule situated in front of the kinetonucleus. The posterior end of the body tapered slightly and the part behind the nucleus frequently contained some chromatin staining granules.

No details of the life-history of this flagellate are known. It is probably a parasite peculiar to the fly. Its presence in the gut of a fly, which has been suggested as the transmitting host of *Leishmania tropica*, and in a locality where Oriental Sore is common, is of importance, for such flagellates, which are frequently nothing more than harmless parasites of the insects in question, can readily be confused with developmental forms of leishmania.

RICHARD GONDER ON THE LIFE-HISTORY OF *THEILERIA PARVA* AND *BABESIA MUTANS*.

[Die Entwicklung von *Theileria parva*, dem Erreger des Küstenfiebers der Rinder in Afrika. Archiv für Protistenkunde, Band xxi. Heft 2, pp. 143–164; Band xxii. Heft 2, pp. 170–178.]

[*Theileria parva* und *Babesia mutans*, Küstenfieberparasit und Pseudoküstenfieberparasit. Ibid. Band xxi. Heft 3, pp. 222–231.]

ROBERT KOCH was the first to recognise East Coast Fever of cattle as a disease *sui generis*, and also to discover the cause in the organism now known as *Theileria parva* (*Piroplasma parvum*). Theiler confirmed Koch's results and was able to separate another parasite, *Babesia mutans*, which very closely resembles *Theileria parva* morphologically, but differs from it in the fact that it is easily inoculable from animal to animal by blood injection, whereas *Theileria parva* cannot be communicated this way. Still later, Theiler has been able to separate another parasite of cattle for which he has proposed the name *Anaplasma marginale*.

In these investigations the author used mostly clean cattle from England and ticks (*Rhipicephalus appendiculatus*) which were either uninfected or infected only with *Theileria parva*, which parasite, unlike the *Babesia*, is unable to pass through the egg of the tick to the succeeding generation.

In the cattle the first parasites are found about twelve days after the ticks have bitten in the form of small round extra-globular structures with single nuclei. They are to be discovered at this stage only in small numbers in the lymphatic glands or spleen. During the following days these forms are also found in the same organs within large mononuclear lymphocytes. The parasites increase in size, there is nuclear multiplication, and finally a breaking up into as many daughter cells as there are nuclei. The host cell may break up before the parasite, so that the latter is liberated, and before final schizogony it may become divided into a number of irregular masses. The merozoites resulting from this schizogony again increase in size and multiply, so that the number of parasites in the lymphatic glands and spleen becomes very large. The author calls these forms, which are asexual, Agamonts, and by schizogony they break up into Agametes, which again grow into Agamonts. After some time the Agamonts, instead of giving rise to Agametes, break up into other forms, which are either extra- or intra-cellular and differ from the Agametes in having a very compact and deeply staining nucleus. These increase in size and give rise to Gamonts, which divide up into uninuclear forms which are young Gametocytes. The young Gametocytes attack the red blood corpuscles, and for the first time parasites appear in the peripheral circulation. It occasionally happens that certain Gamonts enter the red blood corpuscles and there break up into Gametocytes. In this way can be explained the multiplication forms seen in the peripheral blood by some authors. The fact that only Gametocytes are found in the peripheral blood explains the fact that it is impossible to transmit the disease by blood inoculation. In the case of *Babesia* asexual forms occur in the blood, and accordingly diseases caused by these parasites are easily inoculable. A further difference from the *Babesia* infections is that an animal once recovered from East Coast Fever no longer harbours the parasite, while in the *Babesia* diseases the recovered animal remains a reservoir for the virus and may itself suffer from relapses. These relapses, according to the author, are due to a parthenogenetic reproduction of the macrogametocytes, a form of reproduction which does not take place in the case of *Theileria parva*.

As already stated, the first parasites to be noted are the uninuclear extra-cellular forms found in the lymphatic glands and spleen. The nuclei of these young Agamonts are very irregular in structure and possess no nuclear membrane. Within the chromatin mass may be seen one or two deeply staining karyosomes. As the Agamonts increase in size, the nucleus divides successively by a form of primitive mitosis, till, finally, there may be as many as seventy or eighty. The Agamont then divides up into a number of irregularly shaped daughter individuals. Some of these daughter individuals may show a second chromatin body, which according to the author indicates a relationship with the Flagellata. In the case of the intracellular forms, development proceeds in the same manner within the lymphocyte, except that a larger number of daughter cells is produced. However, in these cases it is difficult to be sure that there is not more than a single Agamont in each cell. With the growth of the parasite the cell protoplasm and even the nucleus is destroyed. The characteristic feature of the Agamonts is the irregularity of their nuclei and a marked alveolar protoplasm. The youngest forms measure from ·8–1 μ in diameter, while the full-grown Agamonts may reach 12–15 μ.

After the successive formation of Agamonts for several generations, eventually the Agamonts break up into young Gamonts. Before this takes place various nuclear changes may occur in the Agamonts. This may have to do with the separation of a vegetative from a sexual part of the chromatin. The young Gamonts are distinguished by having a single compact, deeply staining nucleus with a central karyosome. In all cases the schizogony process is irregular, so that the Agamont may break up directly into the young Gamonts or it may first become divided into smaller masses containing several nuclei which again divide up into the Gamonts. The young Gamonts, which have a diameter of ·7–1 μ, grow rapidly, either free or within the lymphocytes, to the adult Gamont with a diameter of 12 μ. The multinuclear Gamont then divides up either directly into Gametocytes or into smaller masses which eventually divide into Gametocytes. Sometimes these smaller masses may enter red blood corpuscles and there break up finally into Gametocytes. In life the newly formed Gametocytes are very motile and may change from a rounded to an elongated form and actively enter the red blood corpuscles.

The latest report on the morphology and behaviour of the forms in the red blood corpuscles is that of Nuttall, Fantham, and Porter. These observers describe an increase in size of the intracorpuscular forms and their motile activity. The author confirms the growth, for the gametocytes increase in size and become either rounded or bean-shaped parasites, or elongated narrow forms. As regards the motility, the author has not found this marked in the intracorpuscular forms. On the contrary, he has seen only very slow movement.

The development described above takes place chiefly in the lymphatic and hæmolymphatic glands, and it is only when asexual multiplication is coming to an end that forms appear in the circulation. As a result of this, it is found that whereas in *Babesia* infections, where multiplication takes places within the red blood corpuscles, there are marked blood changes in the form of Anisocytosis, Basophilia, Polychromatophilia, &c., in the case of infection with *Theileria parva* there is little change in the blood.

As already remarked, inoculation of blood from an animal infected with *Theileria parva* does not convey the disease, as only Gametocytes are thus inoculated, but inoculation of material from the internal organs which contain asexual forms is capable of bringing about infection.

When taken up by the tick the Gametocytes very quickly leave the host cell. The great majority of these perish, many of them being immature. They become rounded and throw out pseudopodia like small Amœbæ. The author is inclined to think that those forms which have several pseudopodia are really degenerating forms. In normal development only one process is thrown out, so that the organism becomes elongated with one pointed end. In the male Gametocytes the nucleus becomes less compact and divides unequally into a larger and smaller part, so that it has some resemblance to a flagellate. The female Gametocyte likewise forms a similar but shorter process, and its nucleus gives off, as a reduction body, part of its chromatin. In this way the male and female Gametocytes become the male and female Gametes. The two Gametes become applied to each other and fusion of the protoplasm takes place. Further reduction of both the male and female nuclei takes place and then fusion of the nuclei occurs. The resulting Zygote is a motile Ookinete, in which there is often formed a small second nucleus which is again evidence of a flagellate affinity. As an Ookinete, the parasite may persist a long time, up to the moulting-stage of the tick. The further development of the Ookinete takes place evidently by its leaving the gut for the tissues of the tick, where it grows into large cysts containing multinuclear protoplasm. Within the cyst there is segmentation into secondary cysts, which again in their turn become broken up into small uninuclear sporozoites having a diameter of 1 μ. These forms occur in the salivary glands and are undoubtedly the stages in which the parasite is injected into the host when the tick fixes. If the larval tick takes up infected blood it is the nymph which transmits the infection, or if the nymph takes up parasites it is the adult tick which conveys the disease. The adult tick never passes on the infection through the egg to the larvæ.

In a comparative study of the two organisms *Theileria parva* and *Babesia mutans*, the one the cause of East Coast Cattle Fever and the other a harmless parasite, the author finds that in the blood there are only very slight morphological differences. As a rule, *Theileria parva* occurs in greater numbers in the blood and does not produce any great change in the red blood corpuscles. In the case of *Theileria parva*, examination of material obtained from the lymphatic glands or spleen shows enormous numbers of the reproducing forms described above, while these forms are not to be found in infections with *Babesia mutans*, which is a typical blood parasite, reproduces in the red cells by simple division, and grows at the expense of the cells, which become very much altered. In both infections cross forms caused by four parasites are occasionally found. In *Theileria* infections the group is a result of the agglomeration of four Gametocytes, while in *Babesia* it is the outcome of a parthenogenetic reproduction of the Macrogametocyte. The Gametocytes of *Babesia mutans* are, on an average, slightly larger than those of *Theileria parva*. The chief differences between the two parasites are these: firstly, the reproducing forms of *Theileria parva* to be found in spleen and gland-juice are not seen in the case of *Babesia mutans*: and secondly, it is only rarely that *Babesia mutans* infects many red cells; sometimes in *Theileria parva* 60–80 per cent. of those cells are infected.

In the case of East Coast Fever, Koch first described peculiar bodies which are to be found in the spleen and glands. These, now known as Koch's globules or Plasma globules, are spherical masses of protoplasm containing one or more chromatin staining granules. They are now shown to be the extracellular schizonts (Agamonts and Gamonts) of the *Theileria parva* and are characteristic of this disease. C. M. W.

THE PARASITIC AMŒBÆ OF MAN.

BY CHARLES F. CRAIG, M.D.,
CAPTAIN, MEDICAL CORPS, UNITED STATES ARMY.

[J. B. LIPPINCOTT CO., Philadelphia and London.]

IN this book of two hundred and fifty-three pages the author has given us a most interesting and readable account of the present position of intestinal amœbiasis of man, with an account of his own results and experience in the examination of these organisms.

In Chapter I. will be found a historic summary of the progress of our knowledge on this subject. Lambl, of Prague, was the first to recognise amœbæ in cases of diarrhœa, though it was fifteen years later that Loesch (1875) studied amœbæ in cases of dysentery and described the organism now known as *Entamœba histolytica*. It was not till Schaudinn's famous publication appeared in 1903 that any clear distinction between a pathogenic and non-pathogenic amœba of man was recognised, though other observers before him had mentioned the existence of such forms. In 1906 Viereck found a third amœba in cases of dysentery, differing in many ways from both *Entamœba coli* and *Entamœba histolytica*, and explaining much of the confusion which had resulted in attempts to differentiate these species. This is *Entamœba tetragena*. In the same year the author described, under the name of *Paramœba hominis*, an organism which has both a flagellate and an amœboid stage in its life-cycle, and which is the cause of diarrhœa in man. In 1904 Prowazek described a species from the mouth to which he gave the name of *Entamœba buccalis*. In addition to these well-defined species, others have been described—*E. tropicalis* (Lesage, 1908), *E. minuta* (Elmassian, 1909), *E. nipponica* (Koidzumi, 1909). It is doubtful if these are valid and species, the same remark applies more certainly to many other supposed distinct amœbæ of the human intestine.

In Chapter II. the General Morphology and Biology of the Amœbæ is considered. The division of the cytoplasm into an ecto- and endoplasm, the character of the nucleus, and the presence of inclusions of various kinds are reviewed. Under the heading of Reproduction the intestinal amœbæ are shown to reproduce in the following ways:—simple division, schizogony gemmation, and reproduction within a cyst. The author states that he has observed Conjugation "repeatedly in *Entamœba histolytica* and *Entamœba tetragena*. It is uncertain just what occurs during the conjugation of organisms of this class, but it is probable that it leads to a rejuvenescence of the vital processes rather than that it is a process of fertilisation." In this case the term conjugation is hardly appropriate, since this is essentially a sexual process when applied to Protozoa, and the author does not mention any nuclear fusion. A better term would be Plasmogamy, which indicates a fusion of the cytoplasm of two or more amœbæ while their nuclei remain distinct. It is not a sexual process, and has been commonly observed in many amœboid organisms.

The amœbæ of man are killed within a few minutes by a solution of quinine (1 to 300) and weak solutions of hydrogen dioxide, permanganate of potash, sulphate of copper, nitrate of silver, argyrol, and dilute acids. Though such agents may bring about the destruction of the amœbæ upon the slide, this is not so readily done in the gut when they have penetrated deep into the tissues.

A chapter on the classification of the Protozoa and the position of the genus *Entamœba* is followed by one on Technique.

The parasitic amœbæ may be studied in the living condition, in stained smears of the material containing them and in stained sections of the fixed tissue. Each of these methods has its advantages, but for diagnosis the examination of the fresh material is the most satisfactory. It is never necessary for diagnostic purposes to stain amœbæ. However, for following out the life-cycle and the nuclear changes stained smears are necessary. The great advantage of stained sections is that they show the exact relation of the amœbæ to the pathological conditions present in the tissues examined.

Before examining a patient for amœbæ it is well to administer a saline cathartic, as this tends to wash the amœbæ from the intestinal wall. One of the most frequent mistakes made in examining material is the use of too thick a preparation. Dilution with normal salt solution or distilled water should be made if necessary. Examination of six or eight specimens with a $\frac{1}{6}$-in. objective and one- or two-inch eyepiece should be made before a negative result is reported. The use of Neutral Red (solution of 1 in 10,000) is often of great service in those cases in which amœbæ are few, as this colours them pink and does not interfere with their movements and does not stain other cells occurring in fæces.

As regards staining, the author says that the best results are obtained by fixation of the wet specimen with osmic acid vapour or sublimate alcohol, followed by staining while the specimen is wet, although air-dried specimens which are afterwards stained are sufficient for ordinary work.

For fixing wet, the best method is to make a film on a cover-glass and while still wet place film downwards in a dish containing the warm fixing fluid, consisting of one part of alcohol and two parts of a saturated solution of corrosive sublimate in normal saline solution. Fixation is complete in a few minutes. This is a good method for subsequent staining by the Iron Hæmatoxylin method of Heidenhain. It is a method which requires considerable experience, but may be said to be the best method we possess for the study of the structural details of amœbæ.

For staining amœbæ with Giemsa's or Wright's stain, dry specimens fixed in alcohol or wet films fixed by exposure to osmic vapour are used.

For staining amœbæ in tissues the author recommends Mallory's method. Small pieces of tissue are hardened in various strengths of alcohol and embedded in paraffin. Thin sections are stained from three to five minutes in a saturated aqueous solution of thionin, differentiated in a 2 per cent. aqueous solution of oxalic acid for $\frac{1}{2}$ to 1 minute, washed in water, dehydrated in absolute alcohol, and cleared and mounted in xylol balsam. With this method the nuclei of amœbæ stain a brownish red, while the nuclei of other cells stain blue. Other methods of staining amœbæ in films and sections are described.

In a chapter on the cultivation of amœbæ, the author gives it as his opinion that it is extremely doubtful if the amœbæ parasitic in man have been successfully cultivated on artificial media. He points out that the amœbæ obtained in such cultures resemble free-living amœbæ like *Amœba limax*, and have resulted from cysts of these amœbæ which have been present in the stool or have contaminated the material used. The author protests against the growing tendency of drawing conclusions regarding the morphology and life-cycle of parasitic amœbæ as observed in man from organisms growing upon artificial culture media. The entire subject of the cultivation of the parasitic amœbæ is in a chaotic condition, and much more work will have to be done before it can be accepted that any of the parasitic species of man have been cultivated."

In Chapter VI. the various amœbæ are individually considered. The author emphasises the fact that it is quite within the power of any careful observer of experience to differentiate between *Entamœba coli* and *Entamœba histolytica* in the living condition, and that such differentiation is essential, not only for diagnostic purposes, but for the subsequent treatment of the case. Details are given of the common occurrence of *Entamœba coli* in the fæces of healthy individuals from various parts of the world and in patients suffering from diseases other than dysentery. *Entamœba coli* consists of a mass of cytoplasm containing a well-defined nucleus, and in a few instances one or more vacuoles. The differentiation between ectoplasm and endoplasm is not well marked. Motility is present, but is very sluggish compared with *Entamœba histolytica*. Under favourable conditions reproduction occurs by simple division and schizogony with the production of eight daughter amœbæ. If unfavourable conditions arise the organisms encyst and eight daughter cells are formed with the cyst, which are liberated under favourable conditions. In size *Entamœba coli* varies from five to fifty microns, but, as a rule, it is seen from ten to twenty microns; in the encysted condition it varies from ten to fifteen microns. In *Entamœba coli* the pseudopodia are short and blunt; in colour it is a peculiar dull grey, while it is exceedingly difficult to differentiate the ectoplasm from the endoplasm, even when the animal is actively motile. The entire cytoplasm in the vast majority of *Entamœba coli* appears to be composed of endoplasm, as the differentiation of ectoplasm is impossible in most instances. During motion the pseudopodia, which are formed by ectoplasm, appear to be somewhat less refractive than the endoplasm and of very slight consistence. The usual absence of vacuoles in this species is emphasised, as also the rare inclusion of red blood corpuscles, even when these are present in the material containing the amœbæ. The nucleus is described as being the most prominent body in the cytoplasm, thus differentiating this parasite from *Entamœba histolytica*, in which the nucleus is generally invisible. In films stained by Romanowsky's stain the endoplasm stains a dark blue or violet, while the ectoplasm is a light blue contrasting with the deep blue staining ectoplasm of *Entamœba histolytica*. The author describes the various methods of reproduction of *Entamœba coli*—simple division, schizogony, and cyst formation. In this respect he has confirmed in every essential detail the results first obtained by Schaudinn. The cystic forms show one, two, four, or eight nuclei, according to the stage of development.

As regards its relation to disease, the author considers that *Entamœba coli* is perfectly harmless, and records experiments made with kittens, wherein he has failed to infect these animals and confirming the similar results of Schaudinn and others. *Entamœba histolytica* is slightly larger than *Entamœba coli*, but the author thinks that size is of doubtful service in diagnosing these species. The average size, however, is from thirty to thirty-five microns. In colour *Entamœba histolytica* has an almost colourless, very refractile ectoplasm, resembling a piece of ground white glass, while the endoplasm is of a very light grey tint, in many instances tinged with green due to absorbed hæmoglobin. This amœba readily engulfs red blood corpuscles, and this may be observed under the microscope. The nucleus, about five microns in diameter, is always very difficult to distinguish or is quite invisible, and is very poor in chromatin compared with the nucleus of *Entamœba histolytica.* A minute karyosome is situated at the centre of the nucleus. The endoplasm generally contains one or more vacuoles. *Entamœba histolytica* is very much more active than *Entamœba coli.* The reproduction of *Entamœba histolytica* takes place by simple division, budding of small daughter cells, and the formation of resistant, thick, capsuled spores measuring three to seven microns in diameter. In this amœba also the author has been able to confirm Schaudinn's observations on its life-cycle. The pathogenicity of *Entamœba histolytica* is demonstrated by the production of dysentery in kittens by its means: this being experimental confirmation of Schaudinn's and Werner's results.

Entamœba tetragena is slightly smaller than *Entamœba histolytica*, is actively motile, and reproduces by simple division and cysts containing four amœbæ. There is an ectoplasm which resembles that of *Entamœba histolytica*, but it is only distinctly visible when the amœba is in motion.

The nucleus is comparatively large and well defined, and is one of the most characteristic features. Externally there is a well-marked nuclear membrane, sharply distinguished from the endoplasm and very refractile: internally there is a large amount of chromatin situated upon the inner side of the nuclear membrane in nodular masses, and distributed throughout the nuclear substance in the form of a network of minute oval or irregular granules: the karyosome is large and has a centriola or centrosome, surrounded by a clear area, and in some instances two or three concentric areas surround the centriola; the latter is spherical in shape and varies in size from a minute dot to a comparatively large spherical mass. This amœba is phagocytic for red blood cells. By experiments similar to those conducted with *Entamœba histolytica* this amœba is shown to be pathogenic also.

As regards the differential diagnosis of these amœbæ, the author remarks that he is convinced that scores of patients have been treated for weeks and months for amœbic dysentery when the only organism present was *Entamœba coli.* It should be remembered that a differential diagnosis of these species does not rest upon the presence of a *single* morphological feature. On page 202 is given a table showing the chief differences between the three common Entamœbæ of man.

A description of *Entamœba minuta* is given. This amœba, named from its small size (12–14 microns), was described by Elmassian from cases of dysentery in Paraguay. It resembles a small *Entamœba histolytica*, but reproduces by simple division schizogony into four daughter cells and cyst formation with the production of four amœbæ. The author thinks that Elmassian may have been working with *Entamœba tetragena. Entamœba nipponica* is probably one of the more common pathogenic amœbæ, as also is *Entamœba tropicalis* of Lesage. *Entamœba undulans* of Castellani is most probably a form of *Trichomonas intestinalis.*

Paramœba hominis was described by the author from a case in the Philippines. It occurs as an amœba measuring from ten to twenty-five microns. It has a distinct nucleus and an ectoplasm. Reproduction takes place by simple division. This goes on for some time, when the amœbæ become enclosed in cysts having a diameter of fifteen microns. The cysts become crowded with small spherical bodies which escape with rupture of the cyst. These small bodies develop flagella and grow rapidly till they have a diameter of ten to twenty microns. At this stage they are pear-shaped organisms with the flagellum rising from the narrow end, while reproduction by longitudinal division occurs. Eventually the flagellum is lost and the organism enters upon the amœbic stage. The organism can be recognised from other amœbæ by the simultaneous occurrence of the various stages in the fæces at one and the same time.

In Chapter VII. is given an account of Prowazek's *Entamœba buccalis* from the mouth. The author has observed it in the United States and the Philippines. Reproduction is by simple division and by schizogony. There is no evidence that this amœba has any pathogenicity, and it is exceedingly doubtful if it has anything to do with caries of the teeth, though it is most frequently encountered in cavities of the teeth.

Amœbæ have been described on several occasions from urine, but it is probable that these are one of the commoner intestinal forms which have gained entrance to the bladder, as occurred in one instance, through a fistula, in the author's experience.

Entamœba miurai, described by Ijima from a peritoneal exudation, is probably not an amœba, while *Entamœba kartulisi* from an abscess in the jaw (Flexner and Kartulis) and *Entamœba pulmonalis* (Artault) are most likely *Entamœba histolytica.*

There is made upon page 43 a statement to the effect that the author was able "to confirm all the essential details of Schaudinn's work upon *Entamœba histolytica* and *Entamœba coli* in air-dried smears of material containing these parasites stained with Wright's modification of the Romanowsky stain, although only after months of study and the examination of many thousand preparations." The author admits that had a wet fixation method been adopted much time would have been saved; but to anyone who has had much experience in staining these amœbæ, especially the cyst forms of *Entamœba coli,* it must be difficult to understand how the dried cyst could preserve any of its details in their original form. The effect of drying the amœbæ and especially the cysts has such a disastrous effect upon the nuclei especially, that it seems to the present writer that it would be quite unsafe to make any conclusions from the appearances thus presented.

To anyone interested in the subject of intestinal amœbiasis this book will be a great help, for, in addition to other matter, it contains an account of the observations of one who has had considerable experience in the examination of these organisms. C. M. W.

A Contribution to our Knowledge of Gall-Sickness (Anaplasmosis of Cattle).

[Dr. A. Theiler, C.M.G., Government Veterinary Bacteriologist.—Transvaal Agricultural Journal.]

The author points out that at the present time gall-sickness in cattle cannot be identified with a certain entity of disease, though he is convinced that at one time there must have been, as there still is, a disease, more or less prevalent, having as its main characteristic a distinct disintegration of liver and bile, with the striking sequel of jaundice. In the course of time other diseases—redwater, rinderpest, and East Coast fever—made their appearance, so that it became difficult to identify that particular disease by its characteristic symptom because it was common to a number of them.

The pathology of abnormal bile production is discussed, and it is pointed out that this may be a result of one or two causes. Either it is caused by toxic substances absorbed from an inflamed intestine, or it is a result of disturbances in the blood-stream, as, for instance, blood destruction.

In 1903 the author found a trypanosome in a disease which the farmer called gall-sickness, and this disease was reproduced by inoculation of blood containing the parasite. Later a spirochæte which caused typical fever reaction and certain blood-lesions was discovered: then a small piroplasm (*Piroplasma mutans*) was found to be connected with gall-sickness. All these diseases were inoculable, but from present knowledge the author concludes that with these inoculations not only were the parasites under observation transmitted, but others as well, particularly redwater and the one to be described in this communication.

The first scientists to study redwater were Smith and Kilborne in America. They described the parasite, *Piroplasma bigeminum*, causing the disease as occurring in two forms—a pear-shaped organism and "peripheral coccus-like bodies" found situated on the margin of the red corpuscles. These bodies occurred only at the beginning of the winter, and they regarded them as a result of the action of cold, which in some way prevented the development of the pear-shaped forms and so gave rise to the mild form of Texas fever. Very few observers have seen these bodies, but Knuth has described them from S. America, and Luhs and Dschunkowsky from the Transcaucasus region.

The author first described these bodies as "marginal points," and though he always suspected their individuality it was only recently that he obtained convincing proof that they represented a distinct parasite. The proof was of this nature. Ten heifers were inoculated in England with the South African strain of redwater. The animals all passed through a redwater reaction. All the animals recovered and were dispatched to S. Africa. Here, in order to further protect, they were again inoculated with redwater strain. The result was that no redwater appeared in the typical time, but after an incubation period varying from twenty-seven to thirty-five days the animals began to sicken and five of them died. The disease did not resemble redwater and lasted much longer. There were no *Piroplasma bigeminum* in the blood, but the disease commenced with the appearance of the coccus-like bodies on the periphery of the corpuscles. They increased in number with the progress of the disease and disappeared with the recovery. These bodies take the typical chromatin stain and could be easily distinguished from the so-called basophile granules which occurred in the same blood.

The term Texas fever of the Americans does not mean the South African redwater alone, but represents two diseases, the second one being the South African gall-sickness. The name *Anaplasma marginale* is given to the new parasite thus separated from redwater. Anaplasmosis resembles redwater, inasmuch as the blood of an animal which has recovered from the disease remains infective; in other words, the immune animal is the reservoir of the virus. Further, the parasite is transmitted by the same species of tick (the blue tick, *Boophilus decoloratus*) and, as a rule, at the same time. The redwater having a shorter incubation makes its appearance first, the gall-sickness appears later. It is for these reasons that it was so difficult to separate the two diseases. The parasite invades and destroys the red blood corpuscles, causing a primary and acute anæmia (oligocythæmia) accompanied by high fever and a degeneration of the large parenchymatous organs.

The parasite is a protozoon which so far has no simile in protozoology, as it consists only of a chromatic substance which takes the typical Romanowsky colour. It is either round or oval in shape. Occasionally round the parasite the red corpuscles do not stain, so that it is surrounded by a paler zone. Reproduction seems to take place by fusion, as partially divided forms are seen. The parasites are usually situated at the periphery of the corpuscle and may sometimes reach over the margin. They multiply rapidly in susceptible animals and invade a large percentage of corpuscles, 40–50 per cent. in severe cases, and may be met with to the number of three or four in each corpuscle. Calves recover easily from the disease and it passes almost unnoticed while they are young. The greatest susceptibility is shown by imported English pedigree cattle, as the morbidity averages about 100 per cent. and the mortality over 50 per cent. of the infected cattle. It is not yet certain to what extent the high mortality is influenced by age. Mixed infections with both *Piroplasma bigeminum* and *Piroplasma mutans* occur.

Clinically, the first symptom is high fever followed by pronounced anæmia and involvement of the digestive tract, resulting in diarrhœa and evacuations of yellow fæces. The urine is distinctly yellow, but never blood-stained. The clinical picture is by no means typical of anaplasmosis. It applies also to cases of redwater where the acute attack is over and the hæmoglobinaria is either absent or has passed unnoticed. Microscopically, the picture of anæmias is identical. The lesions of Anisocytosis and Poikilocytosis, Polychromasia and Basophilia, and the presence of normoblasts and megaloblasts are also found in the sequel of redwater. In redwater, however, *Piroplasma bigeminum* as a rule has disappeared when the above symptoms are noted, while *Anaplasma marginale* persists for a longer time.

Amongst other post-mortem changes due to the anæmia and jaundice, the liver was always found to be enlarged. It had a swollen appearance, was of a yellow colour, and section showed either a brown, yellow, or saffron-yellow tinge. The cut surface was granulated and had a peculiar glossy shine. The organ contained little bile. The gall-bladder contained inspissated viscid dark green bile. The spleen was enlarged in every instance.

Transmission is effected by means of the blue tick, *Boophilus decoloratus*, which also transmits *Piroplasma bigeminum*. In the case of tick transmission the incubation is longer than with inoculation. As the tick is a one-host tick, it follows that the disease passes through the egg, as in the case of redwater, and it is probable that the same tick may transmit both diseases at one time.

Animals which have recovered show a great resistance to reinfection, though there is no complete immunity. The second reaction may be due to a difference in the resistance of the strains used. An animal which has recovered from redwater retains the parasite in its blood, and a subsequent attack of anaplasmosis may cause a recrudescence of the piroplasm, so that both become visible in the blood.

As regards prevention, the practical deduction is to import pedigree stock as young as possible, at least under one year old, if one wishes to save the greatest number possible, both from redwater and gall-sickness. Pedigree stock for South Africa and for Africa generally must have immunity against the two diseases before their importation is accompanied with the best results.

Empirical experience has shown that gall-sickness can be successfully treated. Application of small doses of calomel (1 drachm) followed by Epsom or Glauber's salt (1 lb. dissolved in water) and succeeded by repeated doses of quinine (2 drachms at a time) is recommended. C. M. W.

In the Fourth Report of the Wellcome Research Laboratories of the Gordon College, Khartoum, Dr. Balfour mentions on page 345 *et seq.* that *Anaplasma marginale* occurs in the Sudan, not only in cattle but also in donkeys. In the latter animals it occurs in association with *Nuttallia equi*, the cause of equine peroplasmosis. What is the transmitting host of the Sudan *Anaplasma* is not yet known, but Dr. Balfour thinks that possibly *Rhipicephalus evertsi* will be found to transmit both the *Anaplasma* and the *Nuttallia.*

C. M. W.

III.—DEPARTMENT OF HELMINTHOLOGY.

By R. T. LEIPER, D.Sc., M.B.

THE HOUSE-FLY AS ESSENTIAL INTERMEDIATE HOST FOR A HELMINTH.

[B. H. RANSOM.—Science, vol. xxxiv. p. 690, November 17th, 1911.]

OWING to its habits of frequenting excrement and food the common house-fly has proved a potent factor in disseminating, merely by contamination, the causal agent of several diseases. It has not been thought, hitherto, to act as an essential intermediary.

So long ago as 1861 Carter noticed that in Bombay "every third fly contains from two to twenty or more nematode worms, which are chiefly congregated in and confined to the proboscis, though occasionally found among the soft tissues of the head and posterior part of the abdomen." Leidy mentions the occurrence of this nematode in 20 per cent. of the flies in Philadelphia, and Piana (1896) states that at certain seasons of the year and in certain localities in Italy it may occur in 20–30 per cent. Gordon Hewitt (1909) is convinced that it is very rare in England, as he has only found two specimens out of many hundreds of flies which he has dissected.

This nematode has still more recently been found by Ransom in a considerable number of the house-flies caught at Washington and in Colorado and Nebraska. A series of stages in the development of the parasite was obtained by examining flies at various periods of their growth, and it was soon established that the infection was acquired during the larval stage. As horse-manure is the favourite breeding-place of the house-fly, and it seemed probable therefore that these nematodes represented the larval stage of some parasite in the horse, the stomachs of two horses were examined shortly after death. A large number of adult *Spiroptera* and numerous smaller and variously sized forms were found. Microscopical examination of these latter gave a complete series of stages in the development and growth of a species of *Spiroptera* closely allied to *Spiroptera microstoma*, whilst the smallest and youngest forms corresponded perfectly to those found in infected flies.

The life-cycle is briefly summed up by Ransom as follows:—"A horse infected with the adult worms excretes their embryos in its fæces. These embryos enter the bodies of fly larvæ developing in the fæces from eggs deposited by house-flies. During the development of the fly larvæ and pupæ, the worms with which they have become infested also undergo a process of growth and development, reaching their final larval stage at about the time the flies emerge from the pupal state. The further development of the worm waits upon the swallowing of the infested flies by a horse, in which event the life-cycle becomes completed by the growth of the worm to maturity."

It is pointed out that these observations are of practical value to sanitarians, as the percentage of flies infected in a given locality represents necessarily the minimal percentage of those which have developed in horse-manure. A fuller account of this interesting investigation is promised for a forthcoming Bulletin of the Bureau of Animal Industry, U.S.A. R. T. L.

SHEATHED MICROFILARIÆ IN THE BLOOD OF MAN BY DAY AND NIGHT.

[BAHR, P.—Journal of Tropical Medicine and Hygiene, January 1912.]

IN 1907 Ashworth and Craig drew attention to the occurrence in the blood of a certain number of Philippinos of a filaria embryo, resembling morphologically that of *Filaria bancrofti*, which did not exhibit the nocturnal periodicity so frequently reported in that species. During an expedition to Fiji for the purpose of investigating Dysentery, Dr. Bahr has been able to amply confirm the observations of Lynch and Wilson and others, that in the natives of Fiji there is a similar lack of periodicity. He notes, however, that (*a*) the Fijian microfilaria is, morphologically, identical with *Filaria bancrofti*. (*b*) The parental form is, as far as can be ascertained, identical with *Filaria bancrofti*. (*c*) Both nematodes live in the same tissues and are associated with the same diseases. (*d*) The same mosquito, *Stegomyia pseudoscutellaris*, is an efficient intermediary for *Filaria bancrofti*. The absence of periodicity does not depend, he states, on the habits or any racial peculiarity in the human host, for if a native of India or the Solomon Islands comes to reside in Fiji his microfilariæ retain their habit of nocturnal periodicity ; but when a stranger acquires filarial infection in Fiji the corresponding embryos exhibit no such periodicity.

Assuming that the Fijian filaria is *Filaria bancrofti*, he adopts the suggestion that the absence of periodicity is a partial adaptation to the habits of the intermediary host *Stegomyia pseudoscutellaris*, which feeds only by day, in much the same way as the nocturnal habits of the micro-*Filaria bancrofti* are an adaptation to the well-known habits of its favourite intermediate host *Culex fatigans*.

[ENGELAND & MANTEUFEL.—Archiv für Schiffs und Tropen-Hygiene,
Bd. xv. Nr. 22, November 1911.]

THESE authors note the presence of sheathless and sheathed embryos in the blood of natives in German East Africa. The sheathed forms occur in about 80 per cent. of the cases, and *Filaria loa* is unknown. The sheathed embryo corresponds in morphology and in size with " microfilaria nocturna," differing from the classical description of Manson only in that it occurs also in the blood during the day.

[CONNAL, A.—Journal of Tropical Medicine and Hygiene, January 1st, 1912.]

THE occurrence of sheathed embryos in the blood by day has been noted in West Africa ; but the possibility of infection there with *Filaria loa* has hitherto been offered as an objection to the validity of the records. Recently, however, Dr. Connal, who is well acquainted with the distinctive characters of the embryos of *Filaria bancrofti* and *Filaria loa*, has found, from a detailed study of the blood of natives at Lagos, that out of thirty-one cases the embryos of *Filaria bancrofti* were found during both day and night in five, and during the day only in one. Of special interest is his statement that while embryos of *Filaria perstans* were obtained from the blood both by day and night in fifteen patients, in no less than eleven they could only be found by day, and in eight cases only at night. It is to be hoped that these researches will stimulate workers in various parts of the Tropics to investigate the local incidence of periodicity, using measured quantities of blood. If the results were forwarded to the sectional Editor they would be collated and published in a future issue. R. T. L.

The Trematode Genus *Opisthorchis.*

[Barker, F. D.—Archives de Parasitologie, xiv. pp. 13–61, 1911.]

The author has endeavoured to collect and incorporate into a single paper all the data on the species constituting the genus *Opisthorchis.* He notes that when studied comparatively the different species which constitute the genus immediately arrange themselves into two easily distinguished groups. In one the yolk-glands rarely extend backwards behind the level of the ovary. In the other they extend beyond the posterior testes, and the acini show a distinct break opposite or near the ovary, separating the yolk-glands of either side into a post-ovarial and a pre-ovarial position. The constancy of this division enables the author to form a new genus, which he names *Amphimerus. Opisthorchis noverca,* a rare parasite in man, then becomes *Amphimerus noverca.*

From a comparative study of the original descriptions and drawings of the flukes found by Lewis and Cunningham in the bile-ducts of pariah dogs with those of McConnell's form now called *Amphimerus noverca,* Barker is convinced that they represent two distinct, although perhaps related species, and suggests for Lewis and Cunningham's fluke the name *Opisthorchis caninus.* It should be recalled that the drawings, &c., upon which these determinations of the systematic position rest were made many years ago. Perhaps too much importance is attached to slight differences which now appear in them. Certainly there is still every reason to suppose that McConnell's flukes were relatively immature specimens, the result of accidental infection of man, and that the dog is their proper host. R. T. L.

Bilharziosis in Surinam.

[Flu, P. C.—Centralblatt für Bakteriologie, Bd. lxi. Heft 4–5, 1911.]

From a detailed study of Bilharziosis as it occurs in Surinam in the West Indies, Flu comes to the following conclusions, which, however, differ markedly from those recently arrived at by Looss. In connection with this abstract, reference should be made to the annotation on Professor Looss' paper in Part I of the Journal at p. 20. Flu concludes that the eggs with terminal and those with lateral spine belong to different species of worms: therefore those with terminal spine to *Schistosomum hæmatobium,* those with lateral to *Schistosomum mansoni,* for the following reasons:—

(1) *Schistosomum hæmatobium* has differently shaped eggs from *Schistosomum mansoni.*

(2) In the male of the *Schistosomum hæmatobium* the anterior border of the lateral folds forms almost a right angle with the anterior portion of the body.

(3) The ovaries of the *Schistosomum mansoni* have always a more or less strongly pronounced winding course, a character not described for any of the other species of *Schistosomum.*

(4) The ootype is asymmetrical in relation to the long axis of the animal. The oviduct opens laterally on the ventral side, where also the shell-glands are situated and terminate.

(5) *Schistosomum mansoni* lives, like *Schistosomum japonicum*, in the region of the mesenteric veins; *Schistosomum hæmatobium* predominates in the pelvic veins.

(6) *Schistosomum mansoni* is the cause of an illness whose symptoms correspond fully with those of Katayama disease caused by *Schistosomum japonicum*; *Schistosomum hæmatobium*, on the other hand, gives rise to bladder symptoms which have never yet been observed in *Schistosomum mansoni*.

(7) The pathological-anatomical changes in *Schistosomum mansoni* deviate considerably from those met with in *Schistosomum hæmatobium*, and correspond exactly with those in the Katayama disease.

(8) To this may be added finally the geographical distribution, which, as Sambon remarks, is totally different in *Schistosomum mansoni* from *Schistosomum hæmatobium*. It is in opposition to Looss' view that the lateral spine eggs must be unfertilised eggs, that in different countries—among others Brazil, the Antilles, and in Surinam—we find no other eggs but these.

Flu studied for a long time 15 patients, but never found other than the typical eggs with lateral spine. In these he nearly always found a normally developed ovum and yolk-cells, and in a good many a normal miracidium. A single type of eggs with lateral spine occurred in the uterus and ootype of about 60 females. Flu noted eggs with lateral spine in the uterus of a female found in the veins of the intestine.

V. A. I.
R. T. L.

Camel Filariasis.

[A. S. Leese.—Journal of Tropical Veterinary Science, p. 400, vol. vi., 1911.]
[Mason.—Journal of Comparative Pathology and Therapeutics, December 1911.]

These papers are of interest to medical men on account of the close relationship which obtains between the adult *Filaria evansi* of the Camel and *Filaria bancrofti*, and the similarity of the lesions they induce. The adults of the Camel Filaria occur in the lymphatic glands, pulmonary arteries, in the tissues of the omentum and of the spermatic cord, and also in the vas deferens. The embryos, like those of *F. bancrofti*, are sheathed, and their appearance in the blood is said to be periodic, but this periodicity synchronises, according to Mason's investigation, with elevations of temperature. "Fever lasts for several days, five, six, or seven, according to the severity of the attack, during which period the embryos are easily found in the peripheral blood. The temperature then falls to the normal for a few days and the embryos are, as a rule, no longer found by ordinary examination of blood-films." Leese states that on some days the number of embryos is about the same during the twenty-four hours. On the whole they are slightly less numerous by day than by night.

In both the severe and chronic cases Mason notes a marked enlargement of certain groups of lymphatic glands with a tendency to suppuration and induration.

Leese and Mason agree in regarding the forms found in India and Africa as belonging to the same species, *F. evansi*; but Balfour, apparently owing to some hesitancy in the confirmation of Mason's preliminary note, has recently burdened the embryo with a separate name, "*microfilaria camelensis*."

R. T. L.

CHECK-LIST OF HELMINTHES PARASITIC IN CATTLE.

(*Bos taurus. Buffelus indicus. Bibos indicus.*)

BY R. T. LEIPER, D.Sc., M.B.,
HELMINTHOLOGIST TO THE LONDON SCHOOL OF TROPICAL MEDICINE.

(Species marked thus * are represented in the Collection of the Helminthological Department of the School.)

NEMATODA.

Non-Bursata.

Family I. ASCARIDÆ, Cobbold, 1864.

(1) Genus ASCARIS, Linnæus, 1758.
**Ascaris vitulorum*, Goeze, 1782.

Family II. FILARIIDÆ, Claus, 1885.

(*a*) Subfamily FILARIINÆ, Stiles, 1907.

(2) Genus SETARIA, Railliet & Henry, 1911.
**Setaria labiato-papillosa* (Aless. 1838).

(3) Genus FILARIA, Müller, 1787.
*(?) *Filaria blini*, Caragean & Marotel, 1903.

(*b*) Subfamily ONCHOCERCINÆ, Leiper, 1911.

(4) Genus ONCHOCERCA, Diesing, 1841.
Onchocerca armillata, Railliet & Henry, 1909.
**Onchocerca gibsoni* (Cleland, 1910).
Onchocerca gutturosa, Neumann, 1910.
? *Onchocerca lienalis* (Stiles, 1892).

Family III. DRACUNCULIDÆ, nov.

(5) Genus DRACUNCULUS, Kniphoff, 1759.
**Dracunculus medinensis* (Linnæus, 1758).

Family IV. SPIRURIDÆ.

(6) Genus THELAZIA, Bosc, 1819.
Thelazia alfortensis, Railliet & Henry, 1910.
Thelazia gulosa, Railliet & Henry, 1910.
**Thelazia rhodesi* (Desmarest, 1827).

Family V. GNATHOSTOMIDÆ.

(7) Genus Gnathostoma, Owen, 1836.
Gnathostoma hispidum, Fedschenko, 1839.

Bursata.

STRONGYLES, Looss, 1911.

Family VI. STRONGYLIDÆ, Cobbold, 1864.

(8) Genus Œsophagostomum, Molin, 1861.
Œsophagostomum biramosum (?).
**Œsophagostomum columbianum* (Curtice, 1890).
**Œsophagostomum radiatum* (Rudolphi, 1803).

(9) Genus Chabertia, Railliet & Henry, 1909.
**Chabertia ovina* (Fabricius, 1788).

(10) Genus Agriostomum, Railliet, 1902.
Agriostomum vryburgi, Railliet, 1902.

Family VII. ANCYLOSTOMIDÆ, Looss.

Subfamily Bunostominæ, Looss, 1911.

(11) Genus Bunostomum, Railliet, 1902.
**Bunostomum phlebotomum* (Railliet, 1900).

(12) Genus Gaigeria, Railliet & Henry, 1911.
Gaigeria pachyscelis, Railliet & Henry, 1911.

Family VIII. SYNGAMIDÆ.

(13) Genus Syngamus, Siebold, 1836.
**Syngamus laryngeus*, Railliet, 1899.

Family IX. TRICHOSTRONGYLIDÆ.

(14) Genus Trichostrongylus, Looss, 1905.
? *Trichostrongylus axei* (Cobbold, 1884).
* *Trichostrongylus extenuatus* (Railliet, 1898).
? *Trichostrongylus retortæformis* (Zeder, 1800).

(15) Genus Ostertagia, Ransom, 1907.
**Ostertagia ostertagi* (Stiles, 1892).

(16) Genus COOPERIA, Ransom, 1907.
**Cooperia oncophora* (Railliet, 1898).
Cooperia pectinata. Ransom, 1907.
**Cooperia punctata* (v. Linstow, 1907).

(17) Genus NEMATODIRUS, Ransom, 1907.
**Nematodirus digitatus* (v. Linstow, 1906).

(18) Genus HÆMONCHUS, Cobb, 1898.
**Hæmonchus contortus*. Rudolphi, 1803.

Family X. METASTRONGYLIDÆ.

(19) Genus DICTYOCAULUS, Railliet & Henry.
**Dictyocaulus viviparus*, Bloch, 1782.

PSEUDOSTRONGYLES, nov.

Family XI. EUSTRONGYLIDIDÆ, nov.

(20) Genus DIOCTOPHYME. Collet-Megret, 1802.
Dioctophyme visceralis (Gmelin, 1789).

Family XII. TRICHOSOMIDÆ.

(21) Genus TRICHURIS, Roederer & Wagler, 1761.
**Trichuris ovis* (Abildgaard, 1795).
**Trichuris discolor* (v. Linstow, 1906).

(22) Genus CAPILLARIA, Zeder, 1800.
Capillaria bovis (Schuyder, 1906).

Family XIII. TRICHINELLIDÆ. Stiles & Crane, 1910.

(23) Genus TRICHINELLA, Railliet, 1895.
**Trichinella spiralis* (Owen, 1835).

TREMATODA.

Family I. FASCIOLIDÆ, Railliet, 1895.

(24) Genus FASCIOLA, Linnæus, 1758.
**Fasciola hepatica* (Linnæus, 1758).
**Fasciola angusta* (Railliet, 1895).
Fasciola magna (Bassi, 1875).

Family II. DICROCŒLIDÆ.

(25) Genus Dicrocœlium, Dujardin, 1845.
***Dicrocœlium dendriticum* (Rudolphi, 1819).
Dicrocœlium hospes, Looss, 1907.

(26) Genus Eurytrema, Looss, 1907.
**Eurytrema cœlomaticum* (Giard & Billet, 1892).
**Eurytrema pancreaticum* (Janson, 1889).

Family III. PARAMPHISTOMIDÆ, Fischoeder, 1901.

(*a*) Subfamily Paramphistominæ, Fischoeder, 1901.

(27) Genus Paramphistomum, Fischoeder, 1901.
**Paramphistomum cervi* (Schrank, 1790).
**Paramphistomum dicranocœlium*, Fischoeder, 1901.
**Paramphistomum epiclitum*, Fischoeder, 1901.
**Paramphistomum explanatum*, Creplin, 1847.
Paramphistomum fraternum, Stiles & Goldberger, 1910.
Paramphistomum indicum, Stiles & Goldberger, 1910.
Paramphistomum parvipapillatum, Stiles & Goldberger, 1910.
Paramphistomum scoliocœlium, Fischoeder, 1901.
Paramphistomum siamense, Stiles & Goldberger, 1910.

(28) Genus Bothriophoron, Stiles & Goldberger, 1910.
**Bothriophoron bothriophorum*, Braun, 1892.

(29) Genus Cotylophoron, Stiles & Goldberger, 1910.
**Cotylophoron cotylophorum*, Fischoeder, 1901.

(30) Genus Cauliorchis, Stiles & Goldberger, 1910.
Cauliorchis cauliorchis, Stiles & Goldberger, 1910.
Cauliorchis crassum, Stiles & Goldberger, 1910.
Cauliorchis papillosum, Stiles & Goldberger, 1910.

(*b*) Subfamily Gastrothylacinæ, Stiles & Goldberger, 1910.

(31) Genus Gastrothylax, Poirier, 1883.
**Gastrothylax ægyptiacus*, Looss, 1896.
Gastrothylax compressus, Brandes, 1898.
**Gastrothylax crumenifer*, Creplin, 1847.

(32) Genus Fischoederius, Stiles & Goldberger, 1910.
**Fischoederius cobboldi* (Poirier, 1883).
**Fischoederius elongatus* (Poirier, 1883).
Fischoederius siamensis, Stiles & Goldberger, 1910.

(33) Genus Carmyerius, Stiles & Goldberger, 1910.

**Carmyerius gregarius*, Looss, 1896.
**Carmyerius spatiosus*, Brandes, 1898.
Carmyerius mancupatus, Fischoeder, 1901.

Family IV. CLADORCHIDÆ, nov.

(*a*) Subfamily Stephanopharynginæ, Stiles & Goldberger, 1910.

(34) Genus Stephanopharynx, Fischoeder, 1901.

**Stephanopharynx compactus*, Fischoeder, 1901.

(*b*) Subfamily Gastrodiscinæ, Stiles & Goldberger, 1910.

(35) Genus Homalogaster, Poirier, 1883.

Homalogaster philippinensis, Stiles & Goldberger, 1908.
**Homalogaster poirieri*, Giard & Billet, 1892.

Family V. SCHISTOSOMIDÆ, Looss, 1899.

(36) Genus Schistosoma, Weinland, 1858.

Schistosomum bomfordi, Montgomery, 1906.
**Schistosomum bovis* (Sonsino, 1876).
**Schistosomum japonicum*, Katsurada, 1904.
**Schistosomum spindale*, Montgomery, 1906.

CESTODA.

Family I. ANOPLOCEPHALIDÆ, Blanchard, 1891.

(*a*) Subfamily Anoplocephalinæ, Fuhrmann.

(37) Genus Moniezia, Blanchard, 1891.

Moniezia alba (Perroncito, 1879).
Moniezia benedeni (Moniez, 1879).
Moniezia denticulata (Rudolphi, 1810).
**Moniezia expansa* (Rudolphi, 1810).
**Moniezia planissima*, Stiles & Hassall, 1892.

(*b*) Subfamily Thysanosominæ, Fuhrmann, 1908.

(38) Genus Thysanosoma, Diesing, 1834.

Thysanosoma giardi (Rivolta, 1878)

(*c*) Subfamily Avitellininæ, Gough, 1911.

(39) Genus Avitellina, Gough, 1911.
**Avitellina centripunctata*, Rivolta, 1874.

(40) Genus Stilesia, Railliet, 1893.
**Stilesia globipunctata*, Rivolta, 1874.

Family II. TÆNIIDÆ (larvæ).

(41) (a) *(*Cysticercus*) *bovis*.
*(*Cysticercus*) *tenuicollis*.

(42) (b) (*Cœnurus*) *cerebralis*.

(43) (c) (*Echinococcus*) *multilocularis*.
*(*Echinococcus*) *polymorphus*.

	Families.	Genera.	Species.
Nematoda	13	23	37
Trematoda	5	13	36
Cestoda	2	7	10
Helminthes	20	43	83

Alphabetical List of Helminthes in Cattle.

Nematoda.

**affinis* (*Trichuris*). See *ovis*.
alfortensis (*Thelazia*).
armillata (*Onchocerca*).
axei (*Trichostrongylus*).
biramosum (*Œsophagostomum*).
**blini* (*Filaria*).
bovis (*Capillaria*).
**columbianum* (*Œsophagostomum*).
**contortus* (*Hæmonchus*).
**digitatus* (*Nematodirus*).
discolor (*Trichuris*).
**extenuatus* (*Trichostrongylus*).
**filicollis* (*Nematodirus*).
**gibsoni* (*Onchocerca*).
**gulosa* (*Thelazia*).
gutturosa (*Onchocerca*).
hispidum (*Gnathostoma*).
**labiato-papillosa* (*Setaria*).
**laryngeus* (*Syngamus*).
linealis (? *Onchocerca*).
**medinensis* (*Dracunculus*).
**oncophora* (*Cooperia*).

**ostertagi* (*Ostertagia*).
**ovina* (*Chabertia*).
**ovis* (*Trichuris*).
pachyscelis (*Gaigeria*).
pectinata (*Cooperia*).
phlebotomum (*Bunostomum*).
**punctata* (*Cooperia*).
**radiatum* (*Œsophagostomum*).
retortæformis (*Trichostrongylus*).
**rhodesi* (*Thelazia*).
scutatum (*Gongylonema*).
spiralis (*Trichinella*).
verrucosum (*Gongylonema*).
visceralis (*Dioctophyme*).
**vitulorum* (*Ascaris*).
**viviparus* (*Dictyocaulus*).
vryburgi (*Agriostomum*).

TREMATODA.

**ægyptiacus* (*Gastrothylax*).
**angusta* (*Fasciola*).
bomfordi (*Schistosomum*).
**bothriophorum* (*Bothriophoron*).
**bovis* (*Schistosomum*).
**cœlomaticum* (*Eurytrema*).
cauliorchis (*Cauliorchis*).
**cervi* (*Paramphistomum*).
**cobboldi* (*Fischoederius*).
**compactus* (*Stephanopharynx*).
**compressus* (*Gastrothylax*).
**cotylophorum* (*Cotylophoron*).
crassum (*Cauliorchis*).
**crumenifer* (*Gastrothylax*).
**dendriticum* (*Dicrocœlium*).
**dicranocœlium* (*Paramphistomum*).
**elongatus* (*Fischoederius*).
**epiclitum* (*Paramphistomum*).
**explanatum* (*Paramphistomum*).
fraternum (*Paramphistomum*).
**gregarius* (*Carmyerius*).
**hepatica* (*Fasciola*).
hospes (*Dicrocœlium*).
indicum (*Paramphistomum*).
**japonicum* (*Schistosomum*).
magna (*Fasciola*).
mancupatus (*Carmyerius*).
**pancreaticum* (*Eurytrema*).
papillosum (*Cauliorchis*).
parvipapillatum (*Paramphistomum*).
philippinensis (*Homalogaster*).

*_poirieri_ (_Homalogaster_).
scoliocœlium (_Paramphistomum_).
siamense (_Paramphistomum_).
siamensis (_Fischoederius_).
spatiosus (_Carmyerius_).
*_spindale_ (_Schistosomum_).

Cestoda.

alba (_Moniezia_).
benedeni (_Moniezia_).
*_bovis_ (_Cysticercus_). [_Tænia._]
cerebralis (_Cœnurus_). [_Tænia._]
denticulata (_Moniezia_).
*_expansa_ (_Moniezia_).
giardi (_Thysanosoma_).
*_globipunctata_ (_Stilesia_).
multilocularis (_Echinococcus_). [_Echinococcifer._]
*_planissima_ (_Moniezia_).
*_polymorphus_ (_Echinococcus_). [_Echinococcifer._]
*_tenuicollis_ (_Cysticercus_). [_Tænia._]

Second List of New Genera and Species of Helminthes recorded in 1911.

Agchylostoma ceylanicum, Looss, 1911 (n. sp.), in _Viverricula malaccensis._—N.
Agchylostoma conepate, Solanet, 1911 (n. sp.), in _Conepatus suffocans._—N.
Amphimerus ovalis, Barker, 1911 (n. g., n. sp., t. sp.), in _Trionyx spinifer._—T.
Anomotænia rhinocheti, Johnston, 1911 (n. sp.), in _Rhinochetus jubatus._—C.
Anoplotænia dasyuri, Beddard, 1911 (n. g., n. sp., t. sp.), in _Dasyurus ursinus._—C.
Antorchis, Linton, 1911, n. nom., for _Mesorchis_, Linton, 1910.—T.
Aploparaksis fuligulosa, Solowiow, 1911, in _Fuligula cristata._—C.
Arduenna strongylina, Railliet & Henry, 1911 (n. g., t. sp.), in _Sus scrofa_ dom.—N.
Arhythmorhynchus frassoni (Molin), Lühe, 1911 (n. g., t. sp.), in _Numenius_ spp.—A.
Azygia robusta, Odhner, 1911 (n. sp.), in _Salmo fario._—T.
Bancroftiella tenuis, Johnston, 1911 (n. g., n. sp., t. sp.), in _Macropus ualabatus._—C.
Bucephalus syndosmyæ, Lebour, 1911 (? 1912), sp. inq.—T.
Catatropis liara, Kossack, 1911 (n. sp.), in _Phœnicopterus roseus._—T.
Centrorhynchus aluconis (Müller), Lühe, 1911 (n. g., t. sp.), in _Circus æruginosus._—A.
Cercaria littorinæ-rudis, Lebour, 1911 (? 1912), sp. inq.—T.
Cercaria margaritæ, Lebour, 1911 (? 1912), sp. inq.—T.
Cercaria minor, Lebour, 1911 (? 1912), sp. inq.—T.
Cercaria nsværenensis, Lebour, 1911 (? 1912), sp. inq.—T.
Choanotænia meliphagidarum, Johnston, 1911 (n. sp.), in Meliphagidæ spp.—C.
Davainea himantopodis, Johnston, 1911 (n. sp.), in _Himantopus leucocephalus._—C.
Desmogonius desmogonius, Stephens, 1911 (? 1912) (n. g., n. sp., t. sp.), in _Chelone mydas._—T.
Echinorhynchus brumpti, Blanc & Cauchemez, 1911, in _Erinaceus europæus._—A.
Filicollis anatis (Schrank), Lühe, 1911 (n. g., t. sp.), in Waterfowl.—A.
Hymenolepis fedtschenkowii, Solowiow, 1911, in _Fuligula cristata._—C.
Hymenolepis megarostellis, Solowiow, 1911, in _Fuligula cristata._—C.
Hymenolepis upsilon, Rosseter, 1911 (n. sp.), in _Anas boschas._—C.
Hymenolepis villosoides, Solowiow, 1911, in _Fuligula cristata._—C.

Hyptiasmus arcuatus (Stossich), Kossack, 1911 (n. g., t. sp.), in *Anas clangula*.—T.
Hyptiasmus lævigatus, Kossack, 1911 (n. sp.), in *Fuligula fuligula*.—T.
Hyptiasmus oculeus, Kossack, 1911 (n. sp.), in *Fulica atra*.—T.
Hyptiasmus ominosus, Kossack, 1911 (n. sp.), in *Grus cinerea*.—T.
Hyptiasmus tumidus, Kossack, 1911 (n. sp.), in *Anser anser*.—T.
Microfilaria camelensis, Balfour, 1911 (n. sp.), in Camel.—N.
Necator africanus, Looss, 1911, in *Anthropopithecus troglodytes*.—N.
Nematodirus roscidus, Railliet, 1911 (n. sp.), in *Cervus*.—N.
Notocotyle quinqueseriale, Barker, 1911 (n. sp.), in *Fiber zibethicus*.—T.
Paracotyle caniculæ, Johnstone, 1911 (n. g., n. sp., t. sp.), in *Scyllium canicula*.—T.
Plagiorhynchus lanceolatus (v. Linstow), Lühe, 1911 (n. g., t. sp.), in *Charadrius dubius*.—A.
Rhadinorhynchus pristis (Rud.), Lühe (n. g., t. sp.), in *Scomber* spp.—A.
Spaniometra oculobia, Kossack (Cohn) (n. g., t. sp.), in *Vanellus melanogaster*.—T.
Spirocerca sanguinolenta, Railliet & Henry, 1911 (n. g., t. sp.), in *Canis familiaris*.—N.
Styphlodora bascaniensis, Goldberger, 1911 (n. sp.), in *Bascanion constrictor*.—T.
Telorchis (Cercorchis) robustus, Goldberger, 1911 (n. sp.), in *Cistudo carolina*.—T.
Thysanotænia lemuris, Beddard, 1911 (n. g., n. sp., t. sp.), in *Lemur macaco*.—C.
Trichostrongylus calcaratus, Ransom, 1911 (n. sp.), in *Lepus sylvaticus*.—N.
Trichostrongylus falculatus, Ransom, 1911 (n. sp.), in *Capra hircus*.—N.
Uncinaria polaris, Looss, 1911, in *Vulpes lagopus*.—N.

A. = Acanthocephala.
C. = Cestoda.
N. = Nematoda.
T. = Trematoda.

n. g. = new genus.
t. sp. = type species.
n. sp. = new species.

IV.—PATHOLOGY.

A Method of Introducing a Hollow Needle into a Vein.

[Dr. J. J. Watson.—Journal of the American Medical Assoc., July 1911.]

Anyone who has had occasion to introduce a needle into one of the superficial veins, whether for the purpose of withdrawing blood or for the intravenous injection of some drug, must have been frequently struck with the difficulties that this little operation sometimes presents. The vein very often slips away from the point of the needle and any fluid injected passes to the side of the vein and not into it.

In order to overcome this difficulty, Dr. Watson has invented an ingenious little method by which one can make certain of introducing the needle into the lumen of the vein.

His method is to first tie a ligature around the arm above the point at which the vein is to be punctured. When the vein becomes nicely prominent, he takes a medium-sized sewing-needle, held in a needle-holder or artery-forceps, and transfixes the skin and upper quadrant of the vein. In this way the vein is anchored to the skin. The artery-forceps are then taken off the head-end of the needle and clamped on to the point-end of the same about a quarter of an inch from the vein.

With the forceps in the left hand the needle is gently raised, thus elevating the upper wall of the vein with the overlying skin. The right hand is thus free to insert the injection-needle into the lumen of the vein just below the transfixing-needle. H. B. N.

The Biochemistry of *Bacillus lepræ*.

[F. B. Gurd and W. Dennis.—Journal of Experimental Medicine, vol. xiv. no. 6, December 1911.]

Now that it has been shown that the organism of leprosy can be successfully cultivated, much attention has been given to the production of vaccines to combat the ravages of this dread disease. A certain measure of success has been recorded by several observers, notably by Rost, Williams, &c., but it is evident that much further investigation is required, especially into the minute chemical constituents of vaccines and organisms in general, before a thoroughly trustworthy preparation can be produced, even if the vaccine method prove in the long run to be the most efficacious method of treating leprosy.

The authors of the paper cited above have carried out a very careful and painstaking analysis of the chemical components of the *Bacillus lepræ* in order that a more analytical examination of the immunity producing substances in the micro-organism may be carried out. They put forward the view that even though the results obtained in therapy with injections of killed bacillary suspensions prove efficacious, it will be necessary to test the relative value of extracts prepared in various ways. Incidentally these investigations have shown that, chemically, the *Bacillus lepræ* differs from the *Bacillus tuberculosis* in several important points, thus further differentiating these two organisms which have so many points in common.

The investigations recorded were chiefly to determine quantitatively the amounts of water, fats, lipoids, and proteins present in *Bacillus lepræ,* and incidentally to determine and estimate, as far as possible, the components of certain of these groups and to test their relative toxicity. The amount of water present in the leprosy bacillus varies somewhat, but was found to range between 21·2 and 34·6 per cent. On the other hand, the average water-content of the tubercle bacillus, as given by various observers, is 85·9 per cent. In the determination of fat the dried material was repeatedly extracted with acetone, the residue after evaporation being dried over sulphuric acid. This residue on further investigation was found to consist of neutral fats, free fatty acids, and cholesterol.

The residue after extraction with acetone was treated with boiling chloroform, which, upon distilling off the solvent, permitted the recovery of a small amount of material. This was found to consist mainly of lecithin with minute traces of phosphorus.

The residue after the treatment with chloroform was found to consist of nucleo-proteid, nucleic acid, and ash.

A summary of the composition of moisture free bacilli, as determined by the authors, is shown in the following Table :—

Acetone—soluble extract....................	34·7 per cent. (neutral fats, free fatty acids, cholesterol).
Acetone—insoluble chloroform, soluble extract..	1·7 per cent. (lecithin).
Residue	63·6 per cent. (nucleo-proteid, nucleic acid, ash).

The essential chemical differences between the leprosy bacillus and the tubercle bacillus are the relatively higher water-content of the latter and the presence of comparatively large quantities of lecithin and cholesterol in the former.

Experiments were also carried out to determine the relative toxicity of the bacillary constituents as compared with the whole organism.

From these experiments the authors conclude that the protein portion of the organism represents practically the whole of the toxic body, and, moreover, that, instead of destroying the potency of the toxic property of the bacillus, the freeing of the organism from its fatty covering rendered the toxin more absorbable, so that it acted more quickly and effectively.

H. B. N.

Observations on the Fungi found in Tropical Bronchomycosis.

[A. Castellani.—Lancet, January 6th, 1912.]

So little is known of the condition described as bronchomycosis and of the causative agents of the same, that any work serving to elucidate the condition is indeed to be welcomed.

Dr. Castellani has done much to aid our knowledge of a disease which appears to be of more common occurrence in the Tropics than in temperate climates. It is undoubtedly often mistaken for other affections of the lungs, notably phthisis. In the present paper the author gives the results of his work in identifying the various species of fungi he has been able to isolate from cases of bronchomycosis. He divides them up under five heads—

(1) Those belonging to the genus *Endomyces*.
(2) Those belonging to the genus *Saccharomyces*.
(3) Those belonging to the genera *Streptothrix* and *Actinomyces*.
(4) Those belonging to the genera *Aspergillus* and *Penicillium*.
(5) Non-determined fungi.

He finds that in Ceylon the commonest fungi found in bronchomycosis belong to the genus *Endomyces*. So far he has been able by suitable cultural methods to isolate twelve different species of fungi belonging to this genus, and a very useful Table, showing the differences on culture of these twelve different varieties, is appended. Of cases in which fungi of the genus *Saccharomyces* were found, he has come across two. In both cases the organisms were found to be identical, and corresponded exactly to the type known as *Saccharomyces krusei*. On repeated subculture, however, some mycelial growth was detected, and, as Dr. Castellani rightly observes, it is probable that this fungus will have to be removed from the genus *Saccharomyces*.

Fungi of the genera *Actinomyces* and *Streptothrix* * have been found by the author in some five cases, but, unfortunately, attempts at cultivation failed. He was, however, more fortunate with regard to the fungi of the fourth group, and was able from three cases which he met with to isolate in two of them two distinct varieties of *Aspergillus*, whilst in the third a satisfactory culture of a *Penicillium* was obtained.

Castellani has carried out some experiments to determine the pathogenicity of these various fungi, and has found that several of them are undoubtedly pathogenic. No details of these later experiments are, however, given, but will doubtless form the substance of a further paper when the author has fully investigated this point.

H. B. N.

Government Report on Leprosy in New South Wales in 1910.

The Report, prepared by the Chief Medical Officer of the Government, gives a general survey of all cases of leprosy known to exist in New South Wales, whether detained in the lazaret or living under suitable conditions at their own homes.

It will be seen that 19 cases of the disease were under treatment at the lazaret at the end of the year, whilst since the establishment, in 1883, of an institution for the reception of lepers, 121 cases in all have been received.

Tables are given showing the nationality of all these cases, and it will be found that of these 121 cases, 51 were white people of European descent, whilst the remaining 70 cases occurred among coloured patients.

Interesting features of the Report are the clinical and etiological notes of cases admitted during 1910, together with the surveys of the white patients who remained under treatment at the end of the year. These are accompanied by excellent portraits of some of the cases under view.

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* Included by Castellani in the Fungi.

Various treatments seem to have been used, chiefly chaulmoogra oil in large doses, gynocardate of magnesia, and antileprol; but from the records of the cases it cannot be said that any of these remedies appeared to have much influence on the course of the disease.

The Report closes with a tabular statement of all cases of leprosy recorded in the Commonwealth of Australia during 1910. H. B. N.

On the Biology of Black Mycetoma.

[G. C. Chatterjee.—Indian Medical Gazette, October 1911.]

Brumpt, to whom we are indebted for a classical treatise on mycetoma disease, distinguishes therein two varieties of black mycetoma, which he designates *Aspergillus bouffardi* and *Madurella mycetomi* (Laveran). The former appears to be exceedingly rare, whilst the latter is more often met with and has a wider distribution, and is the form met with in India.

Although the fungus causing this black mycetoma has been shown in each instance to be a well-marked hyphomycete, hitherto all attempts at cultivating it have failed.

From a case of black mycetoma due to invasion with *Madurella mycetomi*, which came under his notice at the Medical College, Calcutta, Dr. Chatterjee has succeeded in cultivating what he believes to be the true black mycetoma. By placing some of the characteristic black grains recovered from the affected tissue on various media, this author was successful in growing the organism. On agar, after incubation for several days, a well-marked growth was obtained, which on examination showed delicate branching mycelial threads which stained readily and showed a nucleus-like structure inside the threads. They were quite unlike the coarse black threads found in the original grains.

Cultivations on various other media were tried. On potato the growth was dry and black, and no moisture whatever was noted. In broth a peculiar growth was noted. When a piece of the old black growth on agar was removed and placed in broth and the whole well shaken and incubated, in a short time numerous small white colonies were found with radiating rays, and all of them adhering to the sides of the glass tube. No diffuse growth was present, nor was there any scum on the surface.

Animal experiments were tried but gave negative results. H. B. N.

The Incidence of Mediterranean Fever in Malta and its Relationship to the Size of the Goat Population.

[J. W. Eyre.—Lancet, January 13th, 1912.]

Dr. Eyre reviews the progress made in the reduction of cases of Mediterranean fever since the adoption by the Military and Naval Authorities of the recommendations put forward by the Mediterranean Fever Commission, whose work and investigations so clearly pointed the way to the effective undertaking of preventive measures. The absolute prohibition of goats' milk as an article of diet took effect in the Services from the beginning of June 1906. The actual case incidence in 1906 was 89 for the first six months of the year, and for the second half of that year 16 only. In 1907, 12 cases were notified: in 1908, 6 cases: in 1909, 10 cases: and in 1910, 3 cases. Previous to the adoption of these preventive measures the average case incidence of Mediterranean fever in the Navy was 240 per annum.

It will, therefore, be clearly seen that, coincident with the discontinuance of the use of an infective food, an abrupt reduction in the number of cases at once manifested itself. In the case of the Military forces an equally satisfactory result has to be recorded. In the last six months of 1906 there were 40 cases, as contrasted with an average of 199. In 1907 the number of cases for the year was 9. In 1908 there were 5 cases; in 1909, 1 case; and in 1910, 1 case.

Dr. Eyre points out that some critics have arisen who assert that this remarkable diminution in cases is not to be ascribed to the prohibition of the use of goats' milk, but to improved sanitary conditions. He rightly shows that such cannot possibly be the case, as no sanitary improvements of any importance have been carried out. A Table is given of the case incidence in various barracks and hospitals in none of which had any sanitary improvements been effected, and the reduction in the number of cases among troops inhabiting these buildings is equally as striking, showing that sanitary improvements did not enter into the case at all.

Amongst the civil population of the island good results are also shown in the reduction of the disease. Such striking results as can be recorded among the naval and military forces are hardly to be expected amongst a civil population, many of them extremely ignorant and, naturally, not living under the same discipline as prevails in the Services, where orders can be promulgated and strict obedience enforced.

Nevertheless, thanks to the gradual education of the more enlightened, coupled with measures taken against infected goats and their owners, the incidence of the disease has been very greatly reduced amongst the civil population, so much so that the number of cases now recorded per annum is only about half what it was in 1906.

By the systematic weeding-out of infected goats by the authorities, the goat population has been reduced from 17,110 in 1907 to 7,619 in 1910.

It is not too much to hope that, provided ample powers are accorded to the Public Health Authorities of the island to deal adequately with the source of the infective material, Mediterranean fever will soon come to be, not one of the most prevalent diseases, but a comparative rarity. H. B. N.

Further Notes on Leprosy Organism.

By H. BAYON, M.D.

After Captain Williams's* exceedingly clear and detailed explanation of the present stand of researches in connection with the cultivation of the germ of leprosy, but little remains to be said except to report on the result of my own experiments.

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* Lecture given at Lond. Sch. of Trop. Med. by Capt. Williams, I.M.S., B.M.S.

On undertaking to investigate for the London School of Tropical Medicine the various claims put forward at different times by different observers who considered they had cultivated the germ of leprosy, I soon noticed that the majority appeared to have only succeeded in getting a streptothrix or a diphtheroid in pure culture from the nodules of lepers. Moreover, that nobody so far had been able to get his claim to have cultivated the germ of leprosy universally accepted, or even acknowledged by the majority of writers on the subject. In other words, the main difficulty did not consist in getting some germ to grow from the nodules of lepers, but in clearly and definitely proving the relationship of the germs so cultivated to the disease itself and showing them not to be accidental contaminations.

To clear up these points we have to rely upon morphology and incidence, animal experiments and serological tests, which in the case of leprosy include "tuberculine"-like reactions.

Morphology and incidence are practically useless in the present case. Acid-fast germs are ubiquitous; no doubt that with sufficient care I could find, by scraping the floor of any room, acid-fast rods in the dust. We all harbour acid-fasts on the surface of our bodies; therefore any acid-fast germ cultivated from one or several cases of leprosy might always be considered as an accidental contamination. These remarks apply also to streptothrix and diphtheroid, and we know, since the experiments of Sanfelice, that the common, everyday *Streptothrix alba* can break up into cultivable acid-fast rods in the animal body. Moreover, though we may be able, with care and experience, to distinguish the germs of one group of acid-fasts from those of another, we are totally unable to do so with absolute certainty so far as regards the individual members of any group. Bovine tubercle germs are microscopically undistinguishable from the human variety, a smegma germ from Marpmanns, acid-fast from urine, and so forth. Incidentally I may mention that over fifty different acid-fast germs have been cultivated and described to date.

Animal experiments promised to give a more satisfactory solution to the problem, till I found that lesions extremely similar to leprosy in the human being could be produced by injecting avian and, in some cases, human tuberculosis into rats and rabbits. Here I may mention that I produced the clinical features of leprosy—*i. e.* nodules in the skin crammed with acid-fasts and nerve-lesions—in a rabbit by an intravenous injection of a pure culture of the smegma acid-fast germ. Therefore animal experiments alone could not be relied upon to give a decisive answer in doubtful cases.

I turned my attention to serological methods. A glance at the literature on the subject showed that time after time workers in this field of research had been unable to get absolutely conclusive results with acid-fast germs. It was quite clear what these scanty results were partly due to. Acid-fast germs in general appear to be encased in an envelope of a fatty or waxy substance, the so-called Nastin of Deycke Pacha. No efficient antigen could clearly be expected if this casing was not dissolved or burst, so as to allow the inner contents of the germ to be acted upon by its homologous antiserum.

Several methods were attempted to achieve this purpose, and at last it was found that, by repeatedly freezing and rapidly thawing and then violently shaking the bacilli, a satisfactory antigen could be produced. This procedure, in connection with successive and graduated dilutions of antigen linked against falling doses of antiserum, enabled me to straightway differentiate from another various acid-fast germs, such as avian and human tubercle, Timothy grass and smegma, &c., by means of the complement deviation test.

I thereupon set to work upon these lines and found that the serum of a leprous patient at St. George's Hospital gave a so-called "group-reaction" with tubercle, human and avian.

From what we see of the leprosy germ in sections, we must come to the conclusion that it must be something akin to human and avian tubercle. And, in fact, Kedrowsky's leprosy germ, which is a sort of *tertium quid* between human and avian tuberculosis, gave a typical, specific, conclusive complement-deviation reaction with leprous serum, quite distinct from that obtained with human and avian tubercle. A bacillary extract from the same germ was repeatedly injected into a leper. It gave a marked rise of temperature, whilst an extract of smegma bacillus was absolutely inert under identical circumstances.

I now took in hand the non-acid-fast streptothrix and the acid-resisting diphtheroid I had cultivated from a leper at this School, injected them into rats and mice. After periods varying from three weeks to several months, they were found to have turned into acid-fast rods which retained some vestiges of their former character. I then injected further rats with these germs, and inoculated other rats with ground-up nodules from lepers. In all these experiments the result was uniform, in fact identical, as you will soon be able to judge for yourselves from the lantern-slides that will be projected on the screen. I must remark that the direct transmission of leprosy from the human being to the rat does not succeed in every case; I had one success in four attempts.

The lesions in rats produced by these various methods are absolutely undistinguishable from those occurring in genuine or spontaneous leprosy of rodents, and are complete analogues of the changes seen in man when leprosy attacks the internal organs.

I do not know what more can be done to show that leprosy is due to a streptothrix, which can be cultivated on appropriate media, in my case placental agar, and that the disease is transmittable to rats, either direct from man or after culture of the germ, and that all stages of the bacterium appear to be infective, even if in varying degrees.

SOME OBSERVATIONS ON THE RÔLE OF THE EOSINOPHILE CELL IN HELMINTHIC INFECTION.

By P. H. BAHR, M.A., M.B., D.T.M. & H. (Camb.).

THE production of eosinophilia is generally regarded as being a response on the part of the organism to helminthic invasion, or a reaction to the toxic products of these parasites. Diminution in the number of eosinophile cells in the circulating blood in an individual known to be the subject of such an infection would appear to indicate one of two things: either the failure of the organism to respond to the toxins produced by the parasite or the death of the parasite itself.

As an illustration of the first of these hypotheses, I will cite three cases of severe ankylostomiasis which came under my notice in Fiji.

In all these cases the eosinophiles were either greatly reduced in number or were entirely absent from the blood. One case recovered under treatment, but the two others were in the last stages of the disease and died. In both these fatal cases large numbers of worms occurred, the majority of which, determined by Dr. Leiper as *Necator americanus*, were found in the small intestines post-mortem.

The first of the three cases was a Solomon Islander, admitted to hospital with anæmia and cardiac symptoms. His hæmoglobin index was 60 per cent., red cells numbered 1,650,000 and white cells 6250 per c.mm. of blood; there was marked poikilocytosis, but in a differential count of 300 cells the eosinophiles were only 2 per cent. The stools were packed with ankylostome ova. He recovered under treatment.

The second case was an Indian in the last stages of the disease. He was intensely anæmic and œdematous: the hæmoglobin index being, as far as could be ascertained, less than 10 per cent. He was in a demented condition, shivering with cold, and constantly passing stools, consisting solely of bile-tinged mucus containing enormous numbers of ankylostome ova. Thus, in one field of the microscope, with a $\frac{2}{3}$-in. lens and No. 2 eyepiece, no fewer than 250 ova were counted. The red cells numbered 1,562,000, the white 10,920 per c.mm. blood; there was marked poikilocytosis. Only one eosinophile was noted in a differential count of 300 leucocytes.

The third case was of an almost exactly similar nature to the preceding. The patient was also an Indian; there was the same œdema, anæmia, and diarrhœa. The hæmoglobin index was less than 10 per cent. The red cells numbered 1,110,000 and the white cells 5000 per c.mm. blood. No eosinophiles were counted in a differential count of 300 cells. The stools were packed with ova.

Certain observations in filarial infection may be cited in support of the second hypothesis which I have stated.

In dissections of lymphatic glands, infected with adult filariæ, great numbers of eosinophile cells, often in a disintegrating condition, were commonly found surrounding the adult filariæ which were coiled up in the cortex of the gland. It is reasonable to suppose that their presence in large numbers in close proximity to the parasite and in the surrounding lymphatic tissue, as evidenced by microscopical sections, was in some way connected with a toxic excretion of the parasite.

In such cases it is not unreasonable to suppose that after the death and complete absorption of the parasite these eosinophile cells would tend to disappear, both from the peripheral circulation and from the gland-tissues.

The following facts may bear out this interpretation :—

The normal eosinophilia (1–2 per cent.) in a European rose to 10 per cent. shortly after a large number of mosquitoes, artificially infected with *Filaria bancrofti*, had been daily fed on his blood. There was no evidence from frequent examination of the stools of an infection with intestinal parasites.

This eosinophilia persisted for eight months, but after a slight attack of orchitis in August of this year, the eosinophilia became normal once more. It is probable that he became accidentally infected with filariæ, for, owing to the hotter weather prevalent at the time, the filariæ had developed with greater rapidity than usual, and were found in the proboscis of the mosquito as early as the eleventh day after infection. No microfilariæ have even been found in the blood-stream, and one is led to conclude that the absence of eosinophilia at the present time is in consequence of, and dependent on, the death of the invading parasite or parasites, to which the attack of orchitis was in all probability due.

V.—PUBLIC HEALTH.

ANTI-TYPHOID VACCINATION[*].

BY R. TANNER HEWLETT, M.D., F.R.C.P., D.P.H.

Evolution of the Method.

So long ago as 1888 Chantemesse and Widal showed that it is possible to protect mice against an injection of virulent typhoid culture by previous treatment with cultures of the typhoid bacillus killed by heat. The same observers made similar observations on guinea-pigs in 1892. Brieger, Kitasato and Wassermann, and Bruschettini a little later confirmed these results, and in 1894 Haffkine, applying the same principle, prepared his anti-cholera vaccine and vaccinated man against cholera. In 1896 Wright in England, and Pfeiffer and Kolle in Germany, prepared their anti-typhoid vaccines by killing broth-cultures of the organism by heat, and soon the inoculation of man with this typhoid vaccine was carried out on an extensive scale by Wright. The considerable reaction attending the injection somewhat limited its use, and some strictures passed on the method after the South African war almost caused a cessation of anti-typhoid vaccination in the British Army and led to investigation by several committees. In 1903 the Royal College of Physicians of London approved the principle of anti-typhoid vaccination and its application to man, and asserted that it reduced susceptibility to the disease and was accompanied by no direct danger, and in 1904, as the result of 20,000 inoculations by Wright, the College of Physicians repeated its endorsement of the method. In the same year the Council of the Royal Army Medical College and a Commission appointed by the Army Council also gave anti-typhoid vaccination their approval. A German Commission in 1905, an American one in 1909, and a French one in 1911 have all reported favourably on anti-typhoid vaccination. The French Commission's general conclusion runs as follows[†]:—

"There are grounds for recommending the voluntary employment of anti-typhoid vaccination as a rational and practical method of diminishing, by a sensible proportion, the frequence and gravity of typhoid fever in France and in the French Colonies. This recommendation is addressed to all whose profession, whose usual or accidental methods of alimentation, whose daily or frequent association with the sick or with bacillus carriers, expose them to direct or indirect contagion by the bacillus of typhoid fever."

Nature of the Vaccine.

A number of different forms of the vaccine have been prepared, of which the following are the principal:—

1. Living but attenuated bacilli (Castellani). A living virus is generally considered to give the best protection, but obviously can have only a limited use.

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[*] For good summaries on the whole subject see Leishman, "Harben Lectures," Journ. Roy. Inst. Pub. Health, July, August, and September 1910; Schereschewsky, Public Health Rep. no. 66, Pub. Health and Marine Hosp. Service, U.S.A.

[†] Bull. de l'Off. Int. d'Hyg. Pub., April 1911, p. 631.

2. Killed cultures. The cultures may be killed :—
    - *a.* By heat (Wright, Kolle and Pfeiffer).
    - *b.* By antiseptics, *e.g.* phenol (Bassenge and Mayer, Niesser and Shiga).
3. "Sensitised" dead bacilli. The cultures are killed by heat and the bacilli are then saturated with typhoid amboceptor by soaking in an anti-typhoid serum, the excess of which is afterwards removed by centrifuging and washing (Besredka, Netter).
4. Vaccines prepared with pulverised bacilli (Friedberger and Moreschi).
5. Vaccines prepared with bacillary extracts, autolysed cultures, &c.
6. Typhoid endotoxin, prepared by the Macfadyen method, has been found by the writer to possess powerful immunising properties, which are lasting, and the fluid causes a minimum of reaction *.
7. Vaccines prepared by chemical means (treatment of cultures with alkalies, &c.).

Of all methods, the second is the one which has been most extensively employed.

In order to prepare an active vaccine, it was formerly considered necessary to employ a virulent organism. Wassermann, Pfeiffer and Kolle, however, insist on the use of cultures which have a *maximum aridity for typhoid amboceptor*, and this bears no direct relation to virulence ; and Leishman and Harrison, therefore, adopting this principle have made use of non-virulent cultures having good absorptive power, and find these to produce as high an immunity as virulent cultures. The lower the temperature at which a culture is killed, and the shorter the heating, the better. Leishman and Harrison now heat only to 53° C. Many little details have to be attended to in order to obtain a vaccine of maximum protective power ; for instance, if the lysol be added while the fluid is warm, much of the protective power is destroyed. By the adoption of non-virulent cultures and by giving a reduced dose, the amount of reaction following the vaccination is very much less than formerly was the case. The vaccine should not be used until three weeks after the date of preparation. Two inoculations should always, if possible, be administered : a first of 500,000,000 bacilli, and a second, about ten days after the first, of 1,000,000,000 bacilli. With this reduced dosage also the negative phase has by no means the frequency and importance attached to it by Wright ; in fact, Leishman states that in his later series of inoculations no evidence of its occurrence could be found. Vaccination should not be practised on those under the age of 7 years.

Results of Anti-Typhoid Vaccination.

A number of statistics relating to the value of anti-typhoid vaccination has been published. Leishman gives the following summary of the incidence and mortality from enteric in India in twenty-four "test-units." Total number under observation 18,483 (on arrival) –19,314 (at date of report) ; average period under observation, 20 months ; number uninoculated 8936, number inoculated 10,378 ; case incidence of enteric per 1000, uninoculated

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* Proc. Roy. Soc. Lond., B, vol. lxxxiv. 1911, p. 49.

30·4±1·23 *, inoculated 5·39±0·48 ; case mortality per cent., uninoculated 16·9, inoculated 8·9.

In the 'Report on the Health of the Army for the Year 1910' (p. 38, vol. lii.) anti-typhoid vaccination in the Indian army is discussed at some length. On December 31, 1910, the European army in India numbered 71,623, of whom 58,481, or 81·7 per cent., had been inoculated. Of these 10,141 had been inoculated once, 39,971 had been inoculated twice, and 8369 more than twice. Of the inoculated men, 57,796 had not had enteric fever, and 685 had had enteric fever. Of the uninoculated men, 11,634 had not had enteric fever, and 1508 had had enteric fever.

In 1910, 335 cases of enteric fever occurred. Of these, 187 were inoculated men and 148 were uninoculated men. Twenty-two deaths occurred among the inoculated and 24 deaths among the uninoculated. The ratio per 1000 of strength of admissions for enteric fever among the inoculated was 3·19, and the corresponding ratio of deaths was 0·37. Among the not inoculated the admission ratio per 1000 of strength was 12·72, and that for deaths was 2·06. As regards case mortality, the percentage figures are 11·23 for the inoculated and 16·89 for the not inoculated. The compilers remark :—"In whatever way we look at this question, we are confirmed in the conclusion that the case in favour of inoculation against enteric fever is much stronger than some critics of the procedure would have led us to think." Doubtless factors other than anti-typhoid vaccination have conduced to the welcome reduction of enteric fever in India ; but recognising this the above figures admit, I think, of but one interpretation, and are a striking testimony to the value of anti-typhoid vaccination. There is no very definite evidence as to the duration of the protection afforded by anti-typhoid vaccination, but it probably cannot be regarded as lasting for more than two years.

However successful anti-typhoid inoculation may be in the prevention of typhoid fever, it must be regarded as only one of the measures for combating the ravages of this disease, and it is only by the adoption of general measures of hygiene and improved sanitation that the disease will be stamped out. To illustrate what improved sanitation will do to stamp out typhoid fever, the death-rate from typhoid fever for England and Wales may be quoted. This per 100,000 of population in 1871–80 was 33 ; in 1881–90, 20 ; in 1891–1900, 17 ; in 1901, 16 ; in 1910 it had fallen to 4·5 !

[Public Health Reports, vol. xxvi. no. 50, December 15th, 1911, U.S.A. Public Health and Marine Hospital Service.]

Pellagra as observed in Barbados.—A short account by Acting Assist.-Surgeon P. G. Smith of cases observed by him in the Out-patient Department of the General Hospital and at St. Michael's Almshouse, Bridgetown.

The patients all give a history of failure in appetite, loss of weight, lassitude, vertigo, burning sensation in stomach, and, in most instances, diarrhœa. This sort of case rarely improves under treatment, and within a few weeks or months they can be found in the wards at the Almshouse.

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* This means that the figure calculated from the data (30·4) statistically is subject to a correction of plus or minus 1·23.

Later, when serious mental symptoms develop, it becomes necessary to admit them to the asylum for the insane. Physical examination shows marked anaemia, pulse rate increased and weak, temperature normal or slightly subnormal. Skin feels hard and dry, and symmetrical pigmented patches are found over the knuckles, backs of hands, wrists, elbows, ankles, and fronts of legs. These patches are scaly, bran-like, and can sometimes be brushed off, exposing apparently healthy under-skin, which rapidly becomes again pigmented. Lips and gums somewhat spongy: tongue red and swollen, and along its edges may be seen a few pigmented spots—in size from a pin's head to a small pea; in some places the tongue appears denuded of epithelium. There may also be seen on the tongue buccal mucous membrane, and on roof of mouth numerous small vesicles filled with a clear serum, which break and leave small, regular-shaped ulcers. It is noted that in a majority of the cases there was no history of syphilis, no evidence of heart lesion or pulmonary disease, and the genito-urinary system was normal. After admission to the Institution, in a majority symptoms outlined above become exaggerated. Pigmentation becomes more marked, the whole alimentary tract becomes involved. There is a most profuse secretion of a thin frothy saliva dribbling from mouth; vomiting of cerebral type now sets in, vomited material consisting of a pale frothy fluid containing shreds of gastric mucous membrane. There is also a wasting diarrhœa pale and yeasty in character, extremely offensive, and accompanied by griping pains over abdomen. The cases end with the development of mental symptoms requiring more or less restraint, and at this stage, in some cases, there appear on palms and soles wine-coloured splotches, upon which form blebs containing clear serum and at times becoming gangrenous. Of more than 40 cases observed in a period of five months, the author gives a concise account of six, selected as being typical and in no instance having a suspicion of syphilis. In three of these cases the sprue tongue and diarrhœa were present and marked; in the other three not so well marked. They all had, besides, the pigmentation characteristic of pellagra. The author states "this condition can best be described as an acute disease with a tendency to recurrence, the direct cause of which is yet to be established, and characterised by slow onset, lassitude, burning sensation in epigastric region, sore mouth, ptyalism, vomiting, and diarrhœa, with well-marked pigmentation of skin over joints of fingers, wrists, elbows, and ankles. In the majority of cases there are manifest later in the course of the disease, nervous and mental symptoms, indicating rapid fatal termination." Dr. Smith notes that Hillary, in 1766, describes "a diarrhœa alba or white flux" . . . . in which "the stools are whiter than is usual in jaundice, without the yellowish appearance in the skin and eyes," and without "any pain in the region of the liver and bile-ducts." The literature in the Colony on this subject is very meagre, but the author thinks "it is quite possible to conclude that the disease which Hillary described is the same as seen to-day, modified to some extent by racial changes and difference in mode of living." As to etiology there are no available statistics, but it appears that the disease is commoner in females than in males, the proportion being almost two to one. It occurs principally between the ages of 25 and 60, although numerous cases both above and below these ages have been observed. It is much more prevalent among the blacks than the whites, and is confined almost entirely to the poorer elements in both classes. Regret is expressed that there are no facilities in the Colony for the proper pathological study of the condition. Necropsy reveals stomach normal in size, gastric mucosa stripped in numerous areas, leaving muscular coat very thin and the blood-vessels exposed. Small intestines very pale, mucosa stripped in large areas, and at several points patches of acute congestion. Large intestines show no pathological change. The liver pale, large, and soft. Spleen normal in size, very dark and soft, easily torn. Other viscera have appeared normal. Dr. Smith continues :—“As the prevailing disease in this Colony presents all the symptoms ordinarily described in both pellagra and sprue or psilosis, the pertinent questions are : Is this disease pellagra or is it psilosis? Is it modified pellagra or is it modified psilosis? Certainly there are found the unmistakable sore mouth and diarrhœa of sprue, and at the same time the characteristic pigmentation of pellagra. A majority of medical men in the Colony say it is pellagra, others hold that it is sprue. The late Dr. Cuthbert Bowen suggested that it be called ‘Psilosis pigmentosa.’ Whatever it may be called, it is the opinion of the entire medical fraternity of the Colony that the disease is a grave one, is rapidly spreading among the poorer classes, and that radical measures must be adopted for stamping it out.” Dr. C. H. Lavinder (who investigated pellagra in Italy with Sambon) appends a note, in which he states :—“In the opinion of most writers Hillary undoubtedly described and differentiated for the first time the disease to which in 1880 Manson applied the name ‘Sprue.’ Since Hillary's time there have been apparently but very few publications on Sprue in Barbados. At present there exists in that island a disease concerning the nature of which doubts have arisen. At the national conference on Pellagra, Columbia, S.C., in 1909, Manning described this disease and mentioned an official report made by him on the subject. Manning's description is quite similar to the one given in this paper, but he also speaks of extensive gangrene as a common occurrence in his cases. The discussion and case notes of the above paper would certainly lead to the inference that there are at least some cases of pellagra in Barbados. There are mentioned, however, some features inconsistent with pellagra. Notable among such inconsistences are, first, the character of the skin lesions.” Dr. Lavinder objects to “pigmentation,” declaring that dermatologists class the pellagrous eruptions among the erythemas, though he says “there is one stage of the eruption when pigmentation is present”—and especially non-pellagra like—“the scaly bran-like appearance, &c.” described by Dr. Smith. “The pale yeasty stools are characteristic of sprue rather than of pellagra. The cerebral vomiting is unlike both sprue and pellagra. Dr. Smith calls the disease acute, but from these recorded observations of his, acute is hardly the word applicable. Nothing likewise is said as to the periodic seasonal recurrence of acute symptoms characteristic of pellagra. Nor is any great stress laid on phenomena referable to disorder of central nervous system, involvement of which is a prominent feature of pellagra.”

General agreement may readily be given to Dr. Lavinder's criticism, but pigmentation generally follows on the erythematous eruption of pellagra, and it would appear that in Dr. Smith's cases, when seen by him, the illness was already well established.

H. S. Stannus, in an able and careful paper recently read before the Society of Tropical Medicine and Hygiene, described, for the first time, cases of pellagra in Nyasaland. The symptoms, with some minor exceptions, and course of the disease were definitely those of pellagra as known in Egypt and Italy, and described by Sandwith and Sambon respectively. Dr. Stannus said :—“The character of the exanthem as observed in natives is distinctive, and, I believe, does not resemble any other affection among them. Commencing as a deepening in colour, the skin (normally a dark chocolate) becomes a dead black, at the same time slightly raised: this is quickly followed either by the appearance of fine wrinkling over the affected patch, and desquamation takes place with the production of dirty greyish-white *branny* scales, or the surface becomes covered with shining, glazed, cracked patches of epidermis, or again blebs may form, more especially where the skin is softer." This agrees with Dr. Smith's "scaly bran-like appearance," and suggests that Dr. Lavinder is perhaps mistaken in stating such an eruption to be non-pellagrous.

Low, who had observed cases in Barbados, has declared that they were pellagra.

Sandwith, after a consideration of the symptoms, physical signs, and course of the disease, together with photographs of cases occurring in Barbados, finds the disease identical with the pellagra of Egypt, and believes that it is sometimes, in Barbados, confused with sprue. The writer has seen cases in Barbados with the scaly pigmented skin lesions described by Smith and Stannus, but with none of the characteristic mouth lesions and diarrhœa he has since become familiar with in sprue.

May not Bowen's psilosis pigmentosa" and Acting Assistant-Surgeon Smith's cases be pellagra *and* sprue? Hillary described sprue in Barbados in 1766: no reason can be given why sprue should have disappeared since then, its etiology is quite unknown. Likely enough, its incidence has increased recently. So exact and brilliant an observer as Hillary does not describe pellagra, strong presumptive evidence of its non-existence in Barbados at the time when he was seeing and noting his cases of "Apthoides chronica." It is more likely, then, that Dr. Smith's cases, as already suggested, were pellagra and sprue, than "the disease which Hillary described ..... modified to some extent by racial changes and difference in mode of living." The etiology of pellagra is still unknown. Sambon's hypothesis of a protozoon parasite transmitted by *Simulium* at present holds the field as the only one likely to reward research. Sambon's investigations of pellagra in Southern and South-Eastern Europe have, up to date, strengthened this hypothesis.

In other parts of the world, also, pellagra and Simuliidæ are found together. Sambon points out the necessity of looking for cases in children as revealing the endemic centres of the disease—the places where infection occurs. It has long been known that "pellagra" occurred in Barbados: of late it has been persistently declared that no Simuliidæ occur in the island, though no entomologist has recorded this statement so far. The island is small, thickly populated, extensively cultivated, and free of forest and scrub; the roads are many and good; the soil porous limestone; and in only one part of the island is there anything like a stream suitable for the breeding of *Simulium*—accepting as universally true the statements in the text-books as to the habits of this fly,—and for some part of the year this stream is simply a series of small shallow pools connected with each other by the merest trickles. In private communications, the writer is informed that the very great majority of pellagra cases come from the districts in the neighbourhood of this stream, but that *Simulium* is not found there. This, however, is not vouched for by an entomologist. A large part of the field-work in the island is performed by women, and pellagra in Egypt, Italy, and Spain is a disease of the field labourer.

Dr. Smith, in his paper, notes the greater frequency of pellagra in women than in men—an observation recorded before by others. An entomological survey of the island of Barbados would be an easy matter and of great value in the elucidation of the etiology of pellagra, and perhaps of sprue also. The entomology of the island had given for two centuries the clue to the transmission of malaria. It had always been noted that yellow fever and malaria occurred in Trinidad, but yellow fever only in Barbados. When Manson first put forward the mosquito theory of the transmission of malaria, Barbados could have shown which mosquitoes did not harbour parasites, and perhaps have saved Ross much weary and fruitless work with the wrong mosquitoes. It is not wildly improbable, to say the least, that a study of the insect fauna of this small, yet proud, and most ancient of all British Colonies will reveal important facts leading to exact knowledge of the etiology of pellagra.

H. M. H.

VI.—CLINICAL MEDICINE.

A Case of Sarcoma simulating Tropical Disease.

By GEORGE C. LOW, M.A., M.D., and
H. B. NEWHAM, M.R.C.S., L.R.C.P., D.P.H.

Cases of tuberculosis, malignant disease, and other ordinary temperate climate maladies are quite frequently to be met with in the Tropics, and very often when they do occur they simulate some of the tropical diseases very closely and so make a correct diagnosis by no means easy. Of these, malignant disease, and especially sarcoma, is generally one of the worst offenders, though perhaps on the whole the cosmopolitan disease, syphilis, is responsible for more mistakes than any. The following notes of a young man who came from Uganda illustrate some of these points very well and show how, even with the recent laboratory methods of diagnosis, it may be impossible to reach a definite conclusion during life.

Mr. Q., 31 years of age, a planter on a rubber estate between Kampala and Jinja in Uganda, consulted one of us (G. C. L.) on November 7th, 1911, complaining of fever and loss of weight. On his way down to the coast from Uganda he had seen Dr. Johnson at Nairobi, the latter prescribing for him and strongly advising him to come straight home to England at once.

History.—The patient had had a somewhat varied and hard tropical experience. Beginning in Natal he lived there five years, then had a trip home, and then went to British East Africa, passing from there to Uganda. He stayed out in Africa for eight years without coming home, his work during that time being varied, but lately he had been employed on a rubber estate in Uganda. His family history was, on the whole, quite good, a brother, however, had died of sarcoma.

Previous illnesses.—As a boy, patient had always been healthy and quite strong. In Natal he did not suffer from any illnesses. Four years ago he had enteric fever in Nairobi. On recovering from this he went to Usoga (a part of Uganda) and while there contracted malarial fever. He then returned to Nairobi and two months afterwards got another attack of malaria, this eventually ending in Blackwater Fever (July 1909). Since that date he had no further attacks of the latter complaint and only a little acute (? malarial) fever. Never dysentery nor syphilis—gonorrhœa once (February 1911); never tick-fever. Had passed through the sleeping-sickness zones in Uganda, but had not actually lived in them, the rubber plantation being just outside them.

Present illness.—In February 1910 was working on the railway down towards coast. Had one or two attacks of fever. In February 1911 went back to Uganda. In July 1911 he began to find that he was losing strength; low fever (as he described it) then developed and he also began to lose weight. He continued until his weakness prevented him from doing his work and he was then invalided home, staying *en route* at Nairobi, where he saw Dr. Johnson as already described. When here his testicle swelled for four days, but subsided after having been painted with iodine. On the voyage home patient kept much the same, no fever, but he did not pick up and he suffered from rheumatic pains in the hip and the small of the back.

On his arrival in England (November 1911) pain at the lower part of his spine was very marked and also on the right side over the region of the liver. Dressing or any slight exertion tired him very much and he did not feel up for any exertion. He sweated at night a good deal ; there was no cough. He was then passing a lot of urine, 6 or 8 times in the night, and could not hold it for long in his bladder during the day. He presented himself for examination two or three days after reaching England.

State on examination (Nov. 7th, 1911).—Patient was very cachetic-looking, of a dark yellow colour, and very considerably emaciated. Conjunctivæ white and anæmic-looking. Enlarged glands in neck and a mass of glands below and behind left ear. Though carefully looked for, no signs of any eruptions on skin ; the tongue was heavily furred, and manifestly the patient was very weak and feeble and ill. Temperature 100°·6.

Examination of the different systems.

Respiratory system.—No abnormalities could be detected in either of the lungs. The apices, bases, and other parts appeared perfectly normal and no signs of tuberculosis were present, though specially thought of and searched for.

Circulatory system.—The heart was normal in size and the sounds were quite pure, no murmurs being present or any other signs of disease.

Alimentary system.—The tongue was heavily furred, teeth fairly good, no evidence of disease in tonsils, pharynx, or other parts of mouth. Appetite was very poor, but there had been no sickness nor pain after food nor any other signs indicative of disease of the stomach. The liver was not enlarged, its upper percussion limits being at the fifth and sixth ribs, its lower edge just at the costal margin. The spleen was considerably enlarged, extending from the sixth to the twelfth ribs and coming out below the costal margin for fully one inch and a half. It felt very hard and firm, but apparently its surface was smooth and presented no irregularities. The stomach was within its normal limits, and no tumour mass or any other abnormalities could be found in the abdomen.

Urinary system.—The urine, which was of a dark straw-colour, showed numerous prostatic threads (? gonorrhœal) ; it also contained a trace of albumin, but no sugar. Microscopically casts were present (see later).

Nervous system.—The pupils were regular in outline, but unequal in size. The left was smaller than the right, and did not appear to react to light very well. The right reacted to light and during accommodation, but was somewhat sluggish. Eyesight appeared to be good, no deficiency in sight apparently existing.

Knee-jerks : The right was present, but by no means active ; the left was just present, but very feeble. There was no evidence of any cranial nerve paralysis nor of paralysis of the legs, though undoubtedly a certain degree of weakness was present in the muscles of the limbs, especially after exertion.

Hæmopoietic system.—A blood-count gave the following results :—

Red	3,860,000	No malarial parasites seen : a few pigmented leucocytes. No trypanosomes. Three hours' search.
White	5,900	
Hæmoglobin ..	70%	

Differential count :—

	per cent.	Another slide. per cent.
Polymorphonuclears	70	70
Large Mononuclears	5	7
Lymphocytes	20	17
Eosinophile	1	2
Transitional	2	3
Mast Cells	2	1
	100	100

A perusal of the differential and general counts given above shows some anæmia, with the uninuclear cells low, 25 and 24 per cent. only. This of itself should have, of course, excluded tubercle, syphilis, and trypanosomiasis, diseases in which the lymphocytes are high; but sufficient stress, as will be afterwards seen, was not laid upon this fact.

Locomotory system.—Rheumatism and rheumatic-like pains were present in the hips and the small of the back, and the patient could only get up off a couch very stiffly and with difficulty. This condition seemed to be muscular rather than arthritic. The joints about the other parts of the body—knee, elbows, wrists, etc.—were not enlarged, nor were there any inflammatory conditions around them suggestive of gonorrhœal arthritis.

Diagnosis.—As far as the examination had gone then there seemed to be something more than mere malarial cachexia. The neuritic pains might possibly mean undulant fever, or possibly gonorrhœal rheumatism, or, again, it could not yet be definitely stated that he was not a case of trypanosomiasis, so the patient was sent to the wards of the Seamen's Hospital at the Tropical School for further observations and clinical tests.

Further examinations of the blood showed the same low lymphocyte counts, and temperature records indicated that the patient was suffering from a remitting pyrexia. He was kept in bed strictly and during the first few days seemed to improve a little. A careful microscopical examination of the urine, made on November the 8th, 1911, showed that the prostatic threads consisted principally of pus-cells and there were also some of these free. Stained films showed cocci and bacilli, but no definite gonococci were made out. In addition, granular and hyaline casts were numerous, with here and there renal epithelium and white cells. The urine was alkaline, triple phosphate crystals being present.

On November the 10th, 1911, one of the enlarged glands in the neck was punctured: an examination of the juice removed revealed neither trypanosomes nor tubercle bacilli. A Widal's reaction for undulant fever was next tried with a negative result, and examination of the fæces gave no ova of intestinal worms or protozoal parasites. Filarial parasites were likewise absent from the blood.

At this time the idea that the case might be one of malignant disease was entertained, but a suggestion of syphilis having been put forward a course of potassium iodide was prescribed.

Almost immediately after commencing the latter drug the temperature came down to and remained at or below normal, and the patient said he felt distinctly better and had a better appetite. By December the 5th, 1911, there was no doubt about the improvement: the patient was less cachectic-looking, the spleen had distinctly gone down in size, and the pain and stiffness in the back had largely disappeared. This improvement seemed to suggest that there must be a specific element in the case. Casts were still present in the urine, however, and the emaciation practically remained *in statu quo*. Twice between November the 10th and December the 1st, 1911, there had been delirium and a tendency for the patient to become noisy and go off his head.

On December 12th, 1911, came a distinct relapse, the different symptoms coming back again, the condition generally becoming worse, and he looked as if he were going to die. The pigmentation now deepened, suggesting Addison's disease, a gland in the neck began to soften and threatened to break down, while severe vomiting, nausea, and inability to take food also appeared. At this juncture the result of a Wassermann's reaction, which had been done after the apparently beneficial result of the iodide, was returned as being negative, so the diagnosis veered round to tuberculosis with probable implication of the suprarenal glands.

There were points certainly in favour of Addison's disease, for example :—(1) The pigmentation of the skin ; (2) the gastro-intestinal symptoms—the nausea, vomiting, pain in the back, etc. ; (3) the asthenia—weakness, wasting, and the anæmia, the heart, however, though rapid was not irregular ; and (4) ? the evidences of tubercle—the glands in the neck. Failing tubercle the most likely probability was malignant disease involving the suprarenals.

From the date of the relapse (Dec. 12, 1911) patient rapidly went downhill, a persistent vomiting helping to weaken him very much, the cachexia and emaciation deepened, and death took place on the afternoon of December the 16th, 1911, at 3 P.M.

An autopsy was performed by one of us (H. B. N.) on December the 18th, 1911, with the following results :—

Body was emaciated. Many enlarged and hard glands in the neck, especially on the left side.

Lungs. Left healthy, no adhesions. Right firmly bound down by old pleuritic adhesions. Slight congestion at the base.

Heart. Small and flabby. No pericarditis or endocarditis.

Liver. Weight 68 ozs., somewhat bile-stained and studded with numerous nodules obviously of a malignant nature.

Spleen. Weight 30 ozs. Numerous malignant nodules both showing on the surface and in the substance of the organ.

Pancreas. Practically the whole gland invaded with malignant growth and feeling very hard to the touch.

Kidneys. A few scattered malignant nodules found in each kidney.

Suprarenals. Healthy.

Stomach and intestines. Healthy.

A few secondary malignant nodules were found on the pleural surface of the fifth rib on the left side and also on the pleural surface of the fourth rib on the right side. There were also several small malignant nodules on the left side of the bodies of the thoracic vertebræ. No primary malignant growth of bone could be made out.

Microscopic examination of the tumours and viscera.

Sections of the various growths showed that the tumour consisted of a round-celled sarcoma, the metastatic deposits in the liver, spleen, etc. exhibiting the same structure as that in the pancreas and elsewhere. The spleen showed much fibrosis, and old malarial pigment was present in considerable quantity. The kidneys were in a condition of subacute interstitial nephritis, this accounting for the casts and albumin seen in the urine during life.

Remarks on case.—The findings reported above (*e. g.* malignant disease) are of extreme interest in view of the leucocyte counts made during life. The lymphocytes, it will be remembered, were relatively very low; and if this fact had been duly appreciated, tubercle and syphilis should never have been given a thought, because in these a lymphocytosis would undoubtedly have been present. Excluding these two, malignant disease was the most likely. Excision of a gland from the neck at an early stage of the disease might have been helpful. It should certainly have excluded tubercle and trypanosomiasis, but it would not necessarily have indicated round-celled sarcoma, as one could not have been certain that the gland was not only in a state of chronic hypertrophy.

As to the improvement under iodide—and this, as regards the temperature, size of spleen, and other points, was undoubted—one can only say that the coincidence was extraordinary and misleading. Certainly the autopsy bore out the negative Wassermann's reaction, no signs of syphilis being present there either. The subacute nephritis was interesting: could it have developed as an after-result of the Blackwater Fever in July 1909? Such cases have been described by Deaderick in America.

Again, no implication of the suprarenals, though somewhat marked symptoms of Addison's disease were present during life, is also very interesting.

The case in many respects therefore was a most instructive one, and we have reported it somewhat fully, as the explanation of its many puzzling features may help others while struggling with the diagnosis of similar cases in the future.

NOTES ON EARLY STAGE OF AN INFECTION WITH *SCHISTOSOMUM JAPONICUM*.

By A. C. LAMBERT, M.D. Toronto.

A. M. *Family history.*—Good.

Previous history.—Good, no history of hepatitis or dysentery; has had malaria, but not for three or four years.

Present history.—Came to Kiukiang from Shanghai in the summer of 1909, and enjoyed good health until November of that year. Bathed several times in the Yangtzse near the Bund, but desisted after contracting a slight aural discharge. Suffered from a heavy cold in October. Shot a good deal during October and waded about in the paddy-fields and also bathed in some clean ponds in the country; generally wore when out shooting short flannel pants, socks, and boots. Used to come frequently in contact with the muddy waters of the fields and the flood-waters. The highest water-mark of that year was 12 ft. 2 in., some 4 ft. over normal, and a good part of the country round was flooded. His favourite time for snipe-shooting was before breakfast.

On November 11th he complained of not feeling well. Had cold in the head and cough. Was given some aspirin and stayed in the house that day. Next day felt better and went to the office. Felt seedy again on the night of the 12th and temperature was 102°. Stayed in bed next day and was seen by me at 10 A.M., when temperature was normal, but at night rose to 101°, pulse 80. Complained of pains in back and limbs and along the course of the intercostal nerves. Some cough present with scanty expectoration. Percussion showed slight impairment of resonance over left base of lungs and on auscultation fine crepitant rales were heard on deep inspiration, the breath-sounds being diminished over the same area. Treatment consisted of a purge and some aspirin. Temperature normal on the morning of the 14th, but rose to 100°·8 at night. November 15th, morning temperature was 98°, evening temperature 100°, pulse 82. On the afternoon of November 16th urticarial wheals made their appearance on the body and legs. These were accompanied by a good deal of itching and remained out in one situation two or three hours, forming large rings before fading. Cough was still troublesome, so was given an expectorant mixture and mag. sulph. in small and frequent doses. These medicines had little effect on the rash, which continued to appear until the 20th. Salol with quinine aa. grs. 5 was then substituted for the salines. No rash appeared on the 22nd, nor on any of the subsequent days. During all this time the appetite was poor; the tongue thickly coated in the middle with yellowish fur, the edges remaining bright red; his bowels acted only after purgatives or enemas; his temper was irritable and he had little inclination to read or otherwise occupy himself in bed. The morning temperature was always normal: at night it reached its highest point about 7 o'clock and commenced to fall shortly after, reaching the normal again on the following morning about 8. Defervescence was always accompanied by more or less sweating. The cough was persistent and irritating, releasing scanty tenacious mucus in which there was a multitude of organisms, none of which gave promise of being the cause of the illness. However, no cultivations were made. Areas of dulness, which varied from day to day, were discoverable on examination of the lungs. These dull areas could be prophesied as being about to form by hearing some hours before a fine crepitant rale like that preceding the first stage of pneumonia. Pain of a pleuritic nature was not complained of, though a sensation of tightness on taking a deep breath was noticed.

As there was no improvement in the patient's condition after a fortnight, Dr. Barrie, who conducts a sanatorium in the mountains near Kiukiang, was called in consultation. It was decided that the case might be one of incipient phthisis, and treatment at the sanatorium was tried for a month, with the result that the fever disappeared by lysis in about three weeks time and Mr. M. was able to return to duty early in January 1910.

While he was in the mountains he was subjected to both Calmette's and von Pirquet's tests for T.B. Both were negative. An examination of the stools at this time showed nothing abnormal. The blood examination showed a very high eosinophilia. Dr. Barrie obtained 40 per cent. eosinophiles in the third week of the illness. My count, made some days earlier, is as follows:—

	per cent.	
Eosinophiles	34	
Polymorphonuclears	34·5	
Mononuclears	12·5	Malarial parasites absent.
Lymphocytes	18	
Basophiles	1	
	100	

Paracentesis was twice resorted to, because the sounds over the right base led to the idea of fluid being present. None found.

The urine showed nothing abnormal.

The patient lost several pounds weight during his illness, but soon picked them up with exception of about 8 lbs., which amount he has always, since that time, been below normal. During the year 1910 he remained well and was able to attend to his duties, but for many months he was depressed and lacking in mental and physical energy. The little dull area at the right base of the lung remained in evidence for some time and never entirely disappeared, the breath-sounds entering feebly at that point.

His lassitude left him as time went on, and by the autumn of 1910 he was more like himself and was able to indulge in some shooting excursions with benefit to himself. On these occasions, however, as I had come to the conclusion, from seeing other cases of a similar nature, that the cause of the fever lay in the waters in which he had been accustomed to wade and bathe, Mr. M. was very careful to clothe himself in thick breeches, putties, and good boots, through which variety of clothing any water which might reach his skin would be well filtered. He suffered from no fever during the winter of 1910 nor the spring of 1911. He did not gain in weight, but otherwise appeared to enjoy his usual health.

In the early summer of this year my attention was called to an article published in the 'China Medical Journal' by Dr. Logan on the case of a young foreigner who had had attacks of fever similar to Mr. M. and in whose stools there were eventually discovered the eggs of *Schistosomum japonicum*. Beyond the history of bathing and wading in ponds, etc., near the Yangtzse, the type of temperature, and the eosinophilia there was not much in common between the two cases. However, Dr. Houghton, of Wuhu, who saw the case with Dr. Logan, noted the similarity in the various points mentioned and suggested that the cause of these types of fever with eosinophilia might sometimes be infection with *Schistosomum japonicum*.

In July of this year (1911) Mr. M. asked me to attend him for a severe influenzal cold. I asked to see a morning stool free from admixture with urine or disinfectants, and on the top of the fæcal mass there was a little streak of blood-stained mucus which contained the eggs of *Schistosomum japonicum*. A second examination made some days later revealed no eggs, but a third examination made during another attack of nasal catarrh, to which Mr. M. appears to have been rather subject, revealed some more eggs, though this time there was little blood in the mucus. A blood-count made on Sept. 24th, 1911, gives the percentage of cells as follows :—

	per cent.
Eosinophiles	21
Polymorphonuclears	37·5
L. Mononuclears	13·5
Lymphocytes	28
	100

My opinion is that the original fever that Mr. M. suffered from in 1909 was caused by the entry through the integument of the larval form of *Schistosomum japonicum* in certain numbers: that their passage through the tissues to the portal circulation, and perhaps the effects of their metamorphoses therein, caused the rise in temperature, which ceased when the worms had reached maturity, or the system became used to their presence.

On making the discovery of the eggs I informed the patient of it and advised him that while under ordinary circumstances there was not much danger to health to be apprehended, so long as no fresh infection occurred, at the same time it was highly essential that for a considerable period he should live amidst surroundings which should tend to keep his health and spirits well up to the normal. To this end I advised Mr. M. to apply for two years' leave and to proceed to some mountain resort on the Continent where he could get all the outdoor exercise and amusement he required with a minimum risk of catching those common colds to which he is so very subject, and during the exacerbations of which there are always present more eggs in the stools than can usually be found, showing that lowered states of health may have some tendency to increase the passage of the eggs through the mucosa of the bowel or their production by the female worm.

In addition it is perhaps superfluous to point out that acute diseases of the gastro-intestinal tract, the contracting of which might be most prejudicial to ultimate recovery from the schistosomiasis, are exceedingly prevalent on the Yangtzse and elsewhere in China, so that it is in great measure to avoid any such undesired consequences that I have given him the above-mentioned advice.

I would also put forward the suggestion that on his return to China he be posted to a part of the Empire free from endemic Schistosomiasis Intestinalis, and where acute disorders of the gastro-intestinal tract are not common.

The following is a report on the physical condition as shown at the present time. It should be pointed out that the patient is suffering from one of his coryzal attacks, for which due allowance should be made in weighing the symptoms :—

Heart.—Normal.

Lungs.—Diminished freedom of air-entry at both bases, more marked on the right side. Sonorous rales and moist rales heard over the same areas. Expansion good, but causes coughing when limit is reached. The patient has not, during the time he has been under my care, made sufficient use of the peripheral portions of his lungs. Percussion note is resonant, but poor in tone over most of the lung-area. *Sputum* : Owing to the cough an examination has been made of the sputum, which is bronchitic in appearance, fairly abundant in the early mornings, and contains large numbers of *Micrococci catarrhalis,* but no *Bacillus tuberculosis.*

Liver.—No tenderness on percussion or compression. Lower level reaches 1½ ins. below costal margin in the erect, and 1 in. in the prone position. No nodules or thickenings to be felt along the margin. Upper level of dulness reaches to fifth interspace.

Abdominal Organs.—Normal. There is no tenderness to be elicited anywhere throughout the abdomen.

Renal System.—Normal. Urine : Sp. Gr., 1010 : Sugar, none ; Albumen, none.

Nervous System.—Normal. Knee-jerks normal.

Examination of Blood.—Eosinophilia about 20 per cent.

Fæces.—Presence at times of streaks of mucus, sometimes blood-stained, containing the eggs of *Schistosomum japonicum.*

NOTE.—The patient was seen in England, November 1911 ; there was still eosinophilia 11 per cent., but no eggs were found. The patient is apparently in good health. Liver normal size and no abdominal trouble. No blood or mucus was found in the stool.

The case is of special interest in connection with "Urticarial Fever" and also as showing that the severe, and often fatal, sequelæ of infection with this parasite do not necessarily occur. C. W. D.

Amœbic Dysentery.

By A. DUNCAN, M.D., F.R.C.P., Lt.-Col. I.M.S. retd.

When I was a medical student, and for some years afterwards, Dysentery was held to be a single disease. As is well known now, the term "Dysentery" means rather a generic term for more than one disease, differing in their etiology and treatment. Thus under this heading are included the Dysenteries due to various forms of bacteria, and to animal parasites such as the protozoal, for instance. Again, as the Commission sent out in our late South African War to investigate the subject showed, a form of dysentery may arise owing no causal relation to any organism. In this paper I propose to deal with the form of dysentery falling under the second head, namely the Amœbic or Tropical.

Years ago I read the Clinical Articles by Messrs. Councilman and Lafleur in the 'Johns Hopkins Reports,' vol. 2, 1891, and also the article by Councilman in the 'Boston Medical Journal' for July 1892. Since reading these articles I have never, until quite recently, met with a case at all resembling their descriptions. My experience of dysentery was gained chiefly in the Punjab, the United Provinces, and Afghanistan, and in common with those of my service who have served in these parts of the world, it was by no means a small one. I came to the conclusion that such a form of dysentery as therein described did not exist in the Punjab or United Provinces, and in this I was at the time supported by the experience of Surgeon-Major Buchanan, I.M.S., who in a paper written some ten or twelve years ago, I think for the 'India Medical Gazette,' came to the conclusion that the amœbæ were simply concomitants. For against the view of their causal relation we have the facts that many observers in Egypt have failed to discover them; that there is no relation between the severity of the illness and the number of the amœbæ—thus in two fatal cases recorded by Gasser in Algeria the amœbæ were very scanty, whilst in others that did not succumb they were found in large quantities; that they have been found in other diseases, as cholera, typhoid, pellagra, tubercular ulcers of the intestines, subphrenic abscess following typhoid fever, colitis, chronic prostatitis, and chronic enteritis. One has also found them in normal intestines. To this argument, however, is opposed the fact that the amœbæ here found are of a different variety, being smaller in size and with a more purely granular protoplasm; also they do not take up the red blood corpuscles. The disease was said to be characterized by the following peculiarities:—

(1) *Its chronicity and irregular course.*—Now of course in civil practice one meets with cases of chronic dysentery in the dispensaries of India, but with respect to my experience in military hospitals, I cannot recall a single case of chronic dysentery during the whole time I was serving in India.

(2) *Relapses alternating with periods of comparative quiescence.*—This feature I never met. A man who had had an attack of dysentery was by it predisposed to another, but I never saw any relapse properly so called. Lafleur, however, does not put this feature forward, as he states he has never seen a distinct relapse.

(3) *Irregular diarrhœa, which may or may not come on suddenly and at intervals.*—This I never came across in any of my cases.

(4) *The less frequent occurrence of pain and tenesmus.*—As regards Europeans, I never met cases of acute dysentery in which pain and tenesmus did not occur. Amongst natives these symptoms were not so marked naturally, as natives do not complain so much of pain in any disease as Europeans.

(5) *The appearance in the disease of ordinary dysenteric stools at the onset, to be succeeded by a marked increased fluidity of the evacuation.*—I never noticed this characteristic fluidity of the stool.

(6) *Latency and frequent absence of dysenteric symptoms.*—These I never experienced.

(7) *Duration 6–12 weeks.*—I have never met with a case in military practice having this duration. It was an essentially acute disease with a short duration.

(8) *Extreme fatality.*—Now from my military experience I should say that dysentery was amongst the least fatal of diseases.

(9) *Extremely slow convalescence.*—All my patients have been able to return to duty very quickly.

But there are in addition two points in which the cases of dysentery I saw in the Punjab and United Provinces differ from cases of amœbic dysentery. First as regards the post-mortem appearance. My experience here is, I must confess, limited, for two reasons—one being that very few of the dysentery patients died, and the other that in a regiment it is extremely difficult to get a post-mortem. Yet when I did obtain one I never saw the appearances described by Professor Leonard Rogers, of my old service, in his classical paper read at the Annual Meeting of the British Medical Association at Manchester in 1892. This officer described appearances which I have never seen ; Major Buchanan, I.M.S., of the U.P., had also never come across such appearances in that part of India. Rogers notes a characteristic yellow gelatinous material filling up the ulcers. He also notes the entire healthiness of the mucous membrane immediately beyond the raised patches, the raised oval ulcers standing up and projecting above the level of the unaltered mucous membrane around them, thus presenting a great contrast to the depressed, worm-eaten mucous membrane so commonly seen in the ordinary bacterial type of dysentery. Other points of difference in my experience were that in Rogers's cases the lesions are nearly always more marked in the upper part of the large intestine and often limited to this part, thus accounting for the frequent absence of tenesmus and the latent character of the symptoms. There was also a freedom of the sigmoid and rectum from lesions, an involvment of the appendix, and the author shows the process to be essentially an infiltration of the submucous coat and not of the mucous membrane. The second point of difference lies in the method of treatment. Up till quite recently, when there have been reports from America of the good effect of ipecacuanha, the opinion of the American physicians was against the value of ipecacuanha in the treatment of amœbic dysentery. In the cases of dysentery I saw in the Punjab and the U.P. I cannot remember one case of failure of cure by this drug. The American physician, on the contrary, recommends rectal injection. Osler states that "medicines administered internally yield on the whole very unsatisfactory results." Strong, in his article in the latest 'American System of Medicine,' an article that is truly remarkable for its comprehensive grasp of the disease in all its aspects, agrees with this opinion. These authorities recommend rectal injections.

According to Manson, on the other hand, ipecacuanha is the specific for the disease. His cases also in our hospital have not been of the chronic variety.

Until quite recently I had never seen a case of amœbic dysentery as described by the American authors. But in September of this year I was consulted by Mr. T. S. F., æt. 19, Midshipman. This officer, in his medical history, told me he had not passed a solid motion for five years; the loose motions were of the type described in the paper in the Johns Hopkins Report, being mucoid with purulent greyish material, occasionally greenish or dark brown with grey-white masses floating in them embedded in blood-stained mucus. They were also very liquid. Bloody stools were not a marked feature. Periods of remission occurred, but the motions were at no time solid. On board ship at Alexandria, Port Said, and the Naval Hospital, Plymouth, he was treated for amœbic dysentery with rectal injection, but with no good result. When he consulted me I should certainly have ordered him rectal injection of quinine had he not told me that they had had no effect, so I at first gave him izal, which, at the hands of Major Vaughan, of my old service, has been so beneficial in the treatment of dysentery. This drug also had no effect, so I had recourse to Manson's method with ipecacuanha. The result was truly striking, for in eight days the character of the stools had completely changed, and his motions have been normal ever since. The change in his personal appearance was also most marked; he at once began to lose his worn appearance and to assume the aspect of health.

It would thus appear to be the case that, corresponding to what we know concerning bacillary dysentery, there must be more than one variety of amœba causing amœbic dysentery. In bacillary dysentery ipecacuanha, which I have found to be an absolute specific for the disease in India, has not ranked thus highly in other parts of the world. To name only a few. In South Africa, Gordon Watson found ipecacuanha to be useless except in the case of soldiers coming from India. Maberley, as the result of six years' experience, spoke enthusiastically of the value of *Monsonia ovata*. This was tried in the wards of the Seamen's Hospital and proved to be without effect. Again, during the South African War ipecacuanha, according to the experience of Sir Frederick Treves, was of no use. (Here, however, as shown previously, the dysentery was not due to any organism.) In North America simaruba bark has been found to be the most efficacious. In Malay, Braddon states the mortality was least after treatment by rectal injection, and most after ipecacuanha. In Japan, Schenke got the best results from calomel. In Egypt, Sandwith preferred sulphate of copper injection. In the Cameroons, Plehn advocated small doses of calomel. In Java, Van Brero recommends an infusion of the fresh leaves of djamg. Day, of the Niger Coast, finds salines far better than ipecacuanha. Ford states, as the result of four years' experience in the United States and England, that anti-dysenteric serum is *the* treatment for bacillary dysentery.

And so with amœbic dysentery. This disease has been recognised in Calcutta and Bombay, and is there amenable to ipecacuanha. In Bombay the organism was similar to the amœba of Noc, and was not the *Amœba histolytica*. Manson, as we have seen, holds this drug to be a specific for amœbic dysentery. The American authors generally do not either mention it or prefer saline injection of quinine. We may state in conclusion that there would appear to be the following varieties of amœbic dysentery :—

A. The class described in the Johns Hopkins Reports, in which the disease is more or less chronic, not amenable to ipecacuanha, but treated best by rectal injection.

B. The class, much more acute, treated successfully by ipecacuanha.

C. The class, chronic in its course, not amenable to rectal injection, but curable by ipecacuanha.

Finally, I would remark I have never been able to understand why this form of the disease is spoken of as "Tropical or Amœbic Dysentery." I have had no personal experience of dysentery in any tropical country except India, but in India the tropical dysentery most frequently met with is *not* the amœbic form but the bacterial form. In this matter I am at any rate supported by Professor Leonard Rogers, I.M.S., the Professor of Pathology at Calcutta, who states that the most frequent form of the disease in that country is not the amœbic but the bacterial. It cannot either be termed tropical on the view that it only occurs *in* the tropics, for a case has been reported during the last year in a subject who had never been out of England.

THE LEUCOCYTE COUNT AS AN AID TO DIAGNOSIS IN DENGUE FEVER.

BY P. H. BAHR, M.A., M.B., D.T.M. & H. (Camb.).

ASHBURN AND CRAIG (Phil. Journ. Sci. vol. ii. no. 2) draw attention to the leucopenia commonly found in dengue fever, which is progressive, being most marked on the fifth or sixth day of the disease. This they regard as of marked diagnostic importance.

The lowest leucocyte count they record is one of 1200 per c.mm. blood, the highest 4860, the average being 3800. Similar figures have been given by other observers.

Dengue of a severe type, and often accompanied by the most varied symptoms, commonly attacks almost every newcomer to Fiji. The initial symptoms are often so severe as to awaken suspicions of typhoid fever. I saw one case in which the temperature (102°–104° F.) remained raised and intermittent up to the eighth day of the disease. There was marked abdominal pain, tenderness, and profuse diarrhœa. The tongue was very foul, and the patient was delirious. The diagnosis was settled on the sudden subsidence of the temperature and the appearance of a rash. In such a case an aid to diagnosis in the early stages is of the utmost importance.

In four consecutive cases, in which a leucocyte count was made, a marked leucopenia was found, and in every case afforded an excellent guide to diagnosis.

In two cases on the third day of the disease the counts were 1200 and 1870 leucocytes per c.mm. respectively. In the other two cases, on the fourth day of the fever the leucocytes numbered 2184 and 7800 per c.mm., but in the latter case they fell to 1870 per c.mm. on eighth day, after which the temperature became normal.

As regards the differential count. The polymorphonuclears were decreased in number, averaging 50 per cent., but the mononuclear count was high and averaged 10 per cent.

Vedder has found a similar decrease in the number of polymorphonuclears, and an increase in the small lymphocytes in this disease, but no increase of the mononuclears.

In two of the cases quoted the temperature became normal on the third day, in the third on the seventh, and in the fourth on the eighth.

The bradycardia, generally noted after the subsidence of the fever, is another marked characteristic. A pulse of 60 per minute was commonly noted, and in one case it was as low as 46.

Ipecacuanha in Treatment of certain Febrile Conditions.

By C. W. DANIELS, M.B., M.R.C.P., and
H. B. NEWHAM, M.R.C.S., L.R.C.P., D.P.H.

The value of the use of Ipecacuanha in full doses has been long recognised in some cases of Dysentery, and its special value in Amœbic Dysentery has been insisted on by Manson and others. In small doses it has its own value in certain diseases of the liver, and in many conditions where a freer flow of mucus is desired from the bronchial tubes and other mucous surfaces.

In this paper we wish to illustrate its value in febrile conditions associated with dysentery, hepatitis, discharging liver-abscess, and in others possibly remotely related to intestinal or hepatic diseases. The descriptions of the cases in the main are taken from the notes by various house-surgeons. The patients, with two exceptions, were treated in the Albert Dock Hospital.

Group I.—*Febrile conditions associated with dysentery.*—In this group, of which four cases are appended, a much larger number could easily be obtained: it will be noted that the cases are early ones, or that on admission they were in an early stage of a relapse. As under ordinary circumstances the pyrexia at the onset of an attack of dysentery is of short duration, and is frequently absent in amœbic dysentery, the proof that the cessation of the pyrexia was the direct effect of the ipecacuanha is not conclusive, but the uniformity of the course is suggestive. Cases 1, 2, 3, and 4.

Case 1.—A. K. Patient was admitted on June 30th, 1909, with a history of diarrhœa and blood and mucus in the stools, accompanied by abdominal pain. On admission his temperature was 103·4 C. Examination of the blood revealed no malaria, but the stools were found to contain amœbæ.

The heart and lungs were normal. Liver was enlarged and extended one inch below the costal margin. There was some tenderness on pressure in the epigastrium. He was at once placed on a full course of ipecacuanha

His temperature rapidly fell (Chart I.), and, after some small oscillations, finally came to normal on July 12th. On July 4th the tenderness had much diminished and there were no abdominal pains.

It was noted on July 10th that the liver had become considerably reduced in size and all tenderness had disappeared. Stools were still fairly liquid, but with some formed material in them.

The stools continued to improve and became well formed. Patient was discharged on July 30th.

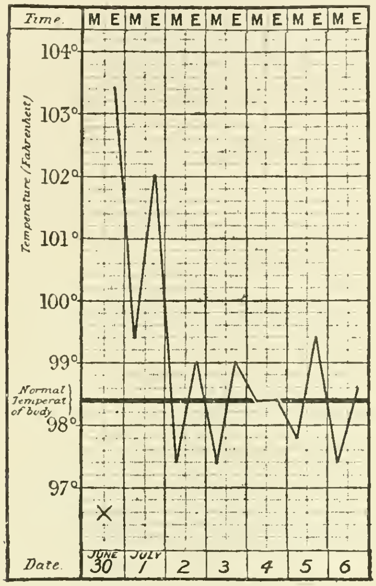

Chart I.—Case 1.

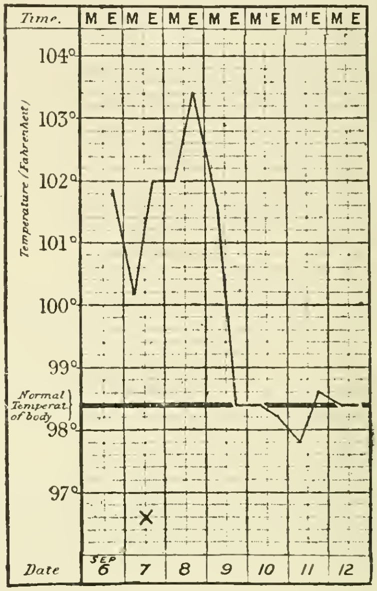

Chart II.—Case 2.

× indicates the day on which Ipecacuanha was first given.

Case 2.—F. D. A., age 25. Admitted Sept. 6th, 1909, with a history of having been ill for five days with diarrhœa, tenesmus, and blood and mucus in the stools.

On admission temperature was 101°·8 C. Patient had six or eight stools a day, containing blood and mucus, and amœbæ were detected therein. On the next day a full course ot ipecacuanha was prescribed, and in two days' time the temperature dropped to normal (Chart II.). The number of stools per diem gradually decreased, the blood and mucus disappeared, and by September 28th the stools were well formed, and patient was discharged on the 30th.

Case 3.—M. F., aged 24. Admitted February 2nd, 1910, with a history of abdominal pain and diarrhœa, with the passage of blood and mucus for the preceding six days. On the day before admission he had 26 stools.

On admission, examination of the abdomen revealed a good deal of tenderness in the left iliac fossa, and slight pain in the right iliac fossa. In the first 24 hours patient passed nine stools containing a good deal of blood and mucus. He was placed on ipecacuanha treatment. On the first four days his temperature was raised to 100° C., but then subsided to normal, and remained so during the rest of his stay in the hospital. The stools gradually diminished in number and the pain and tenderness disappeared, and also the blood and mucus. On March 15th he passed normal stools, and was discharged on March 18th.

Case 4.—M. C., aged 35. Admitted on June 28th, 1907, with a history of having been ill for a few days with headache, shivering, and general pains. No pain in the abdomen or diarrhœa was complained of. It was stated that the night after his illness commenced he had rigors and a high temperature, accompanied by delirium.

Condition on admission.—Temperature 101° C. Liver and spleen not felt. The stools contained much mucus and a little blood. On microscopic examination amœbæ were detected.

The course of ipecacuanha treatment was prescribed. His temperature rapidly fell and became normal on July 2nd, and there was no further fever during his stay in hospital. On July 4th it was noted that his condition was much improved, no blood or mucus being found in the stools, and on July 13th the patient was discharged.

GROUP II.—Cases in which the pyrexia was associated with Hepatitis form an interesting group. There may be associated dysentery or not, and in some cases there has been antecedent operation for liver-abscess, and in other cases symptoms return subsequently, and hepatic abscess is found to be present and require operation; but in all these classes the pyrexia yields to ipecacuanha, if only for a time.

Case 5.—Amœbic dysentery with marked Hepatic enlargement and tenderness.

R. S., aged 27. Admitted November 18th, 1908. Patient gave a history of having suffered from diarrhœa for two and a half months, with blood in the stools for the last three weeks. On admission the stools were liquid, containing blood and sloughs, and amœbæ were found. The liver was markedly enlarged, especially upwards. It extended from the fourth rib in the nipple-line to below the costal margin, and was decidedly tender over the ninth and tenth ribs in the anterior axillary line.

Izal was at first administered, but produced no marked improvement, the temperature ranging up to 101° C.

On November 24th, six days after admission, a full course of ipecacuanha treatment was commenced. Two days later the temperature fell to normal, and remained so during the further progress of the case.

On November 27th, four days after commencing the ipecacuanha treatment, it was noted that all pain had disappeared, and that the area of liver dulness was greatly reduced. Two days later the liver-dulness was noted to be normal. Small doses of ipecacuanha were continued nightly up till January 3rd. Patient was discharged on January 9th, having passed well-formed stools for several days.

Case 6.—Symptoms with fever recurring four months after what appeared to be a successful operation for liver-abscess.

W. F. S., bookstall clerk, aged 33. Admitted to hospital on December 14th, 1910. Had followed his present occupation in England for the last seven years, previous to which he had been a ship's steward trading to the East.

Eight years ago he had dysentery. On returning home had a slight return of the dysentery, but for about six years he has been in excellent health.

Four months ago he began to have shivering fits, profuse night-sweats, and pain in right shoulder.

On admission a diagnosis of liver-abscess was made, and on December 16th an operation was performed, the pus found and drained, and the patient made a complete and uneventful recovery, leaving the hospital cured on January 10th, 1911.

On May 7th he was readmitted to hospital, and stated that up to 17 days previously he had been in good health, but then suffered from headache and pains in the stomach. After a few days in bed he felt better and went to work, but still felt far from well. On May 2nd he was worse and had to give up work and return to bed. Not getting any better he came to hospital and was admitted.

On admission the liver was found to be enlarged, extending from the sixth rib in the nipple-line downwards to three fingers' breadth below the costal margin. In the right lung there was dulness on percussion from the seventh rib downwards in the line of the angle of the scapula. Temperature 103° C.

On May 8th patient was placed on a course of ipecacuanha, and by May 11th the temperature had become normal and remained so (Chart III.).

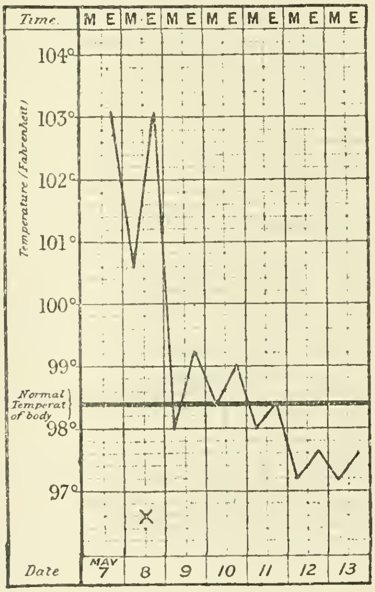

Chart III.—Case 6.

× indicates the day on which Ipecacuanha was first given.

The liver gradually became smaller, and by May 27th, when the patient was discharged, it had resumed its normal size, and all other symptoms had disappeared.

Case 7.—Symptoms with fever return three years after operation for liver-abscess.

R. G., aged 53. Admitted December 4th, 1908. Patient gave a history of pain just above the right hip for four weeks, profuse sweating at nights, right decubitus, cough and slight expectoration. Was operated on for liver-abscess three years previously. On admission the liver was not felt below the costal margin, but dulness extended upwards to the fourth interspace in the nipple-line.

A blood examination gave the figures: red cells 3,500,000, white cells 18,000, hæmoglobin 100 per cent., large mononuclears 9 per cent. Temperature on admission 99°·6, rising to 101° C. the next morning. He was placed on a full course of ipecacuanha. His temperature fell the next day to below normal, and, beyond a slight rise on December 6th to 99° C., never rose above normal during the rest of his stay in hospital. His liver gradually decreased in size, and by the 18th December it was noted that it had resumed its normal size. Patient left the hospital on December 24th feeling quite well.

Case 8.—Pyrexia and symptoms of liver-abscess yield to ipecacuanha, but liver-abscess follows.

L. G., accountant. Admitted to hospital on November 23rd, 1907. Patient gave a history of having contracted dysentery fourteen months ago, followed by frequent relapses, the last attack having been five months previously. He was then treated with rectal injections and magnesium sulphate. The present illness commenced on September 9th, 1907, with fever, vomiting, and abdominal pain, and a few days later the pain developed on the right side over the lower ribs, which was sharp and stabbing in character and increased by deep respiration. On October 12th ipecacuanha was given, which effected some improvement. He had lost a stone and a half in weight since August.

Condition on admission.—Temperature 98°·6 C. The liver extended from the upper border of the fifth rib in the nipple-line to below the costal margin and was tender on pressure. A blood-count revealed 10,000 leucocytes per c.mm. There was a good deal of pain in the right shoulder.

On November 26th ipecacuanha treatment was commenced, and by December 3rd it was noted that the liver was reduced in size, so that its upper border was at the level of the sixth rib in the nipple-line. The pain in the right side was less acute. Patient gradually improved, and was discharged on January 24th, 1908, with very slight pain in the right side and liver apparently at its normal size.

On February 24th, 1908, he was readmitted. The pain in the right side and in the right shoulder had returned and became fairly severe, and patient had already noticed that he became feverish at night.

On admission there was a good deal of tenderness on pressure on the right side below the ribs and also in the right lumbar region. The liver was not made out to be enlarged. The pain gradually became more severe and profuse night-sweats occurred. On March 3rd he was operated on and pus found. This was drained, and patient made an uninterrupted recovery, and was discharged on March 7th, 1908.

These cases were all treated by the method adopted by Sir Patrick Manson for dysentery. Thirty grains of the Pulv. ipecacuanhæ are given the first night, and the dose is diminished by five grains each night till five grains only are taken. Good results as regards the pyrexia can, however, be obtained with smaller doses, though by either method a liver-abscess may subsequently be found.

Case 9.—B. C. Been some years resident in the far East. Had had typhoid, malaria, but no dysentery. Habitual constipation. Had low fever for some weeks, temperature rising to 100° or 101° F., and subnormal in the morning. Much night-sweating. The liver was slightly enlarged and tender. Ipecacuanha in five-grain doses every night was given, and in two days the temperature became normal, the sweats ceased, and the hepatic enlargement and tenderness rapidly disappeared.

Case 10.—G. J. H. A rather doubtful history of dysentery about a year ago. First seen with Blackwater Fever of medium severity (his third attack), with more hepatic tenderness than is usual. The temperature did not fall as the urine cleared, and the pain and tenderness and enlargement of the liver increased. Owing to the weak condition of the patient, doses of ipecacuanha that would not cause vomiting were all that were permissible. Starting with one grain of ipecacuanha every four hours, the dose was gradually increased till there was nausea, by which time he was taking twelve grains a day. The temperature (*vide* Chart IV.) rapidly fell. The liver was less tender and the general condition much improved. In spite of continued administration of the ipecacuanha, after a few days the symptoms recurred and the temperature again rose. The liver was distinctly and unevenly enlarged, and it was decided to operate. A large, recently formed, liver-abscess was found. The patient made a good but slow recovery, interrupted at one stage by an attack of benign tertian malaria, some parasites apparently having survived the Blackwater Fever.

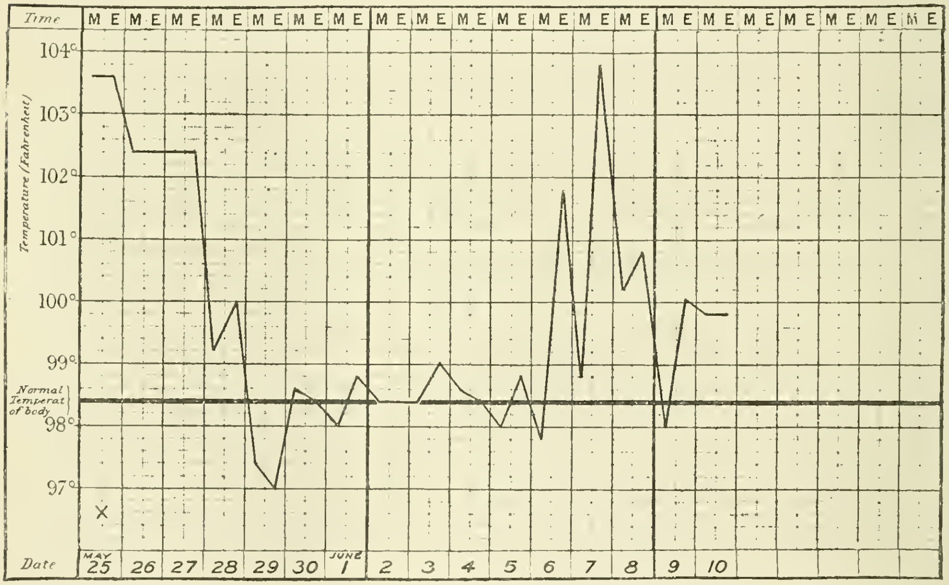

Chart IV.—Case 10.

× indicates the day on which Ipecacuanha was first given.

Group III.—An instructive group of cases illustrating the value of ipecacuanha in liver-abscess which has opened through the lungs or been operated on, but in which recovery is slow and recurrence of the symptoms takes place whilst the discharge continues.

Case 11.—Liver-abscess ruptured through the lungs six days before admission. Rapid improvement when ipecacuanha was administered some fifteen days after the rupture (*vide* Chart V.).

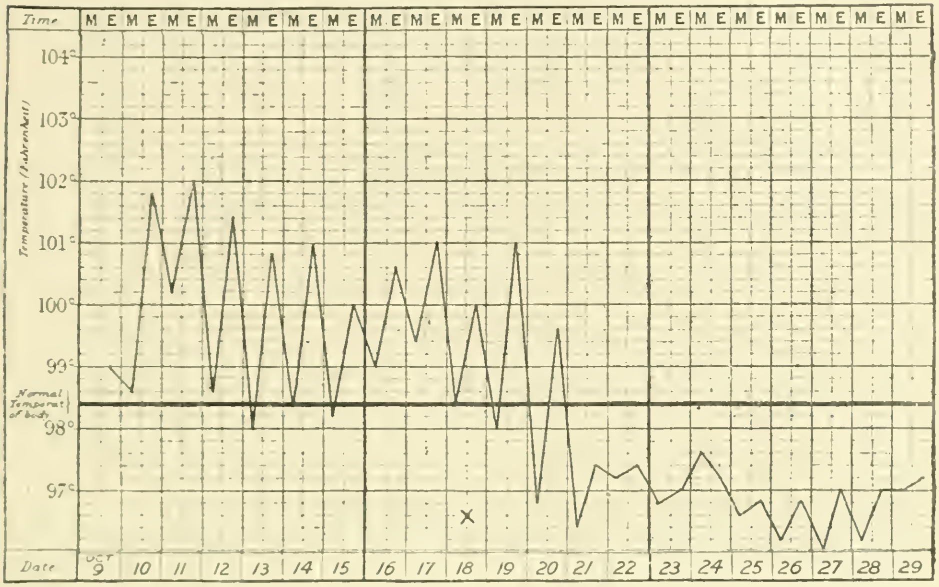

Chart V.—Case 11.

× indicates the day on which Ipecacuanha was first given.

M. V., age 19, seaman. Admitted to hospital October 9th, 1911. Patient stated that he had had malaria two years ago, but had never had dysentery. On October 1st he developed a cold and cough, and three days later had haemoptysis accompanied by a cutting pain in the right side on taking a deep breath and relieved by lying on the right side. He had had no previous lung-trouble.

On admission the patient was pale and anaemic, with a constant cough and dyspnoea. He was bringing up large quantities of frothy sputum containing bright blood. Examination of the chest showed impaired movement on the right side, with dulness to percussion in the right axilla and at the right base behind: breath-sounds diminished at the right base and a few crepitations in the same place.

The liver was enlarged upwards, extending to the fifth rib in the nipple-line, but was not palpable below the costal margin; spleen not enlarged. Temperature ranging up to about 102° at night. No complaint of night-sweats.

On October 18th ipecacuanha treatment was commenced, and by the 21st the temperature became subnormal and remained so during the further course of the case (Chart V.). The sputum gradually decreased in amount, and finally ceased on November 3rd. The patient put on weight and the lung-symptoms cleared up, so that on November 17th the patient was discharged cured.

A glance at the appended Tables will show the rate of diminution in the sputum following the administration of ipecacuanha, and also the progressive gain in weight :—

SPUTUM.

	ozs.		ozs.
Oct. 18th	8	Oct. 27th	4
,, 19th	10	,, 28th	1
,, 20th	8	,, 29th	2
,, 21st	7	,, 30th	1
,, 22nd	6	,, 31st	¾
,, 23rd	4	Nov. 1st	1
,, 24th	6	,, 2nd	½
,, 25th	6	,, 3rd	½
,, 26th	10		

WEIGHT.

	st.	lbs.		st.	lbs.
Oct. 16th	8	11½	Nov. 6th	9	0
,, 23rd	8	3	,, 16th	9	2
,, 30th	8	7			

Case 12.—Liver-abscess ruptured through the lungs ; similar improvement.

G. G., aged 27, mining engineer. Admitted July 19th, 1911.

Past history.—Dysentery in July 1910, for which he was treated in hospital at Bombay for three weeks. Although greatly relieved he was not completely cured, slight dysenteric symptoms appearing from time to time. Shortly after leaving the Bombay hospital he had malaria, and was again in hospital for two months. He arrived in England in December 1910, and then had pain in the right side of his neck and in his right side, and also had an attack of pleurisy. In January 1911 he went to the Gold Coast, but continued to have pain in the right side, and noted that he had an evening temperature and night-sweats. He continued to get gradually worse, and was sent home in June 1911. He arrived in England on July 15th, 1911, and the next day had a violent fit of coughing, and brought up a large amount of what he described as "slimy material, dark pink in colour." Incessant coughing persisted, with the expectoration of a large amount of the same type of material. Patient noted that with the onset of the cough the night-sweats diminished.

On admission, patient looked very pale and extremely ill, and suffering from a persistent cough with the expectoration of typical liver-pus. On examination, temperature was 101°·4 ; slight sweating at night. Stools contained a slight amount of blood and mucus. Liver enlarged upwards to fifth rib in nipple-line, sixth interspace in mid-axillary line, and the lower border extending to two and a half inches below costal margin. The liver was tender on pressure.

On auscultation the respiratory sounds were diminished over liver-area behind with some friction sounds. The amount of expectoration during the first week was 78 ozs. ; during the second week, ending August 1st, 108 oz

On August 3rd ipecacuanha treatment was adopted, and in a few days the cough became somewhat less troublesome and the amount of expectoration less. For the week ending August 8th, five days after ipecacuanha was started, the amount of expectoration had fallen to 67 ozs. On August 8th the patient's weight was 6 stone 13 lbs.

Steady improvement was maintained, the cough and expectoration getting less and less, whilst patient gained in weight.

The amount of sputum for the week ending August 15th was 21½ ozs.; for the week ending August 22nd, 5 ozs.; after which it entirely disappeared.

On August 15th the patient's weight was 7 stone 5 lbs.; on August 22nd, 7 stone 10 lbs.; and on August 29th, 8 stone 1¼ lbs. He was discharged on August 31st.

Case 13.—Liver-abscess, first ruptured through the lungs in Sept. 1909. After this there is the common history of reaccumulation of pus and further ruptures through the lungs. Numerous operations. Abscess located and opened, but neither through the operation-wound nor through the lungs did complete evacuation take place. At intervals the discharge through the lungs recurred. Prompt and permanent relief from the use of ipecacuanha.

R. P., age 32. Admitted October 20th, 1909. Patient stated that four weeks previously he had several teeth extracted, and that three days later he had a sharp attack of coughing with hæmoptysis. Eighteen months ago he had diarrhœa, and again when hæmoptysis started; this time eight to ten motions a day, with griping; but he had not noticed any blood or mucus in the motions.

On examination the liver was found to be greatly enlarged, extending down as far as the umbilicus in the nipple-line on the right side, and to within two inches of the umbilicus in the left parasternal line. No tenderness on pressure. The lung on the left side, over fourth and fifth interspaces, was dull on percussion in front, with feeble breath-sounds. The expectoration was frothy, purulent, and of a dull red "anchovy-sauce" colour. Stools contained amœbæ. On October 27th the liver was explored on both the right and left sides with a needle. No pus was detected in the right side, but on the left side an abscess-cavity was found which yielded some thick, dirty white, creamy pus. This was drained.

After this operation the expectoration diminished from an average amount of 8 ozs. per day to 3 ozs. per diem; but by the 11th of November it was noticed that the liver had increased in size, especially on the left side.

On November 12th the liver was again explored, two inches below the left scapula, and about a pint of pus drained away in 18 hours.

The sputum afterwards got less and less and became colourless, and on December 12th patient was discharged.

After leaving hospital he remained quite well up till December 23rd, when he coughed up a little blood-stained sputum in the morning. He went to bed, and this expectoration of blood-stained sputum continued in the mornings only, and gradually increased in amount, accompanied by slight febrile attacks.

On February 26th he went into a nursing-home, and the liver was again explored with a needle, but no pus found. After this needling patient felt better, but the expectoration did not cease, and gradually increased in amount and became darker in colour.

He was re-admitted to hospital on May 26th, 1910, his liver then being enlarged to one inch below costal margin, but not tender on pressure. Dulness over lower third of left lung, no friction-sounds. Sputum about $\frac{1}{2}$ oz. per diem, flecked with anchovy-sauce-like material.

On June 3rd further exploration with a needle was resorted to, but no pus found, although six punctures were made. Patient remained in fairly good general condition, but expectoration still continued, the amount being about $\frac{1}{2}$–2 ozs. per day.

He left hospital on June 25th, with instructions to report himself from time to time. He remained in much the same condition till the end of September, when he began to have chills, night-sweats, fever, headache, and considerable increase in the amount of expectoration, which became much more purulent in character.

He again came into hospital on October 12th, 1910, with a temperature of 102°, and coughing up some 8–10 ozs. of dark red purulent material a day.

On October 21st the liver was again explored and some pus found in the left lobe. This was drained. The temperature at once fell, and expectoration diminished to about 1–2 ozs. per day, but did not cease.

On November 25th the last operation-wound was opened up and further attempts to get at the seat of the mischief made. Drainage-tubes were inserted and a good deal of liver-pus continued to come away, but still the expectoration did not cease, and patient remained in much the same condition until February 1911. It was then proposed to attempt further surgical measures, but the patient refused, and left the hospital on February 25th, 1911, still discharging pus from the wound in the left side and still bringing up a good deal of liver-pus in the sputum.

The sequel of this case is most important.

The patient had in some way heard that ipecacuanha, taken in large doses, was recommended in the treatment of liver-abscess, and, not being materially relieved in spite of six operations, determined to give this drug a trial in his own case. He therefore procured a supply of Pulv. Ipecac., and started taking it systematically in doses of 10 grains a day. The effect was striking. The sputum rapidly diminished in amount and entirely ceased in about a month. The discharge of pus from the operation-wound in his side also got less and less, and entirely ceased in about five weeks, the wound afterwards healing completely. The patient gained in weight, and now, some eight months later, is in robust health and able to do his work as well as ever—all symptoms having entirely disappeared.

On January 17th, 1912, he still remains in good health: there is no expectoration and no signs of return of abscess. Now and then he has a slight pain in the region of the incision, and when this occurs he again takes a few doses of ipecacuanha.

The Treatment of Tetanus.

[Kintzing.—New York Medical Journal, December 23rd, 1911.]

The author proposes to revive the carbolic acid treatment of tetanus, a method devised first by Bacelli in 1892. Under such a procedure Kintzing has had five consecutive recoveries, while two other cases treated by colleagues in a similar manner also were saved from death. He proposes the following technique for the preparation and giving of the carbolic :—A solution of pure phenol is made by dissolving the deliquesced crystals in ten volumes of sterile water. Of this ten per cent. solution ten drops, equalling practically one grain of pure deliquesced phenol, is the full adult dose. Before injection this requires dilution with sterile water, as otherwise soreness, necrosis, and suppuration may follow. Kintzing took five drops, half the above-mentioned dose, and diluted it with sterile water to the capacity of a twenty-five or thirty minim syringe and injected this into one buttock, the remainder of the dose being immediately injected into the other buttock, in an exactly similar way. In other instances the entire dose, if properly diluted (to forty minims), has been given, however, without any bad results following.

In fully developed cases of tetanus a second dose is administered one hour after the first, and then at intervals of three hours, the intervals being increased as improvement manifests itself. In some of the cases this was very marked after the third or fourth dose.

During the administration of the drug the urine has been carefully watched to see if the characteristic smoky colour develops; but so far Kintzing has never been obliged to withdraw the remedy either for this or any other contraindication. G. C. L.

Carrion's Disease, or Verruga Peruana.

By CARLOS MONGE, M.D. Lima.

As defined by Carrion, "this disease is a pyrexia accompanied by anaemia. It is endemic and is always characterised by wide-spread pains, terminating in a general eruption of a polymorphic character and definite evolution and generally persisting, uninfluenced by treatment, for a long time."

Distribution.—Verruga is found only on the western slope of the Peruvian Andes. It occurs there in certain small towns—Quebrada de Huaylas, Santa Eulalia, San Matteo, &c.—situated in deep defiles branching off from the main valley. These towns are very hot and badly ventilated, because the defiles in which they are built run in a direction at right angles to that of the prevailing winds. The disease is never acquired naturally below an elevation of about 2800 feet, or above 9000 feet; nor does it originate, even in the endemic districts, in the main valleys where the air circulates freely, although it may be common in the small towns in the neighbourhood and perhaps only a few kilométres distant.

Etiology.—Very little is known on this subject. In addition to its distribution one other salient fact is known, namely, that most cases occur during the summer months when the rivers in the districts mentioned are in flood. At that time all sorts of insects abound, and the flooded districts become hot-beds of malaria.

For a long time I have been convinced that earthquakes, very common occurrences in these districts, have occasioned small epidemics. It is more reasonable, I think, to regard this coincidence not as one of cause and effect, but as implying an opportunity for the spread of infection owing to the over-crowding of the towns consequent on occasions of this sort.

Neither race, age, nor sex is a bar to infection. Strangers, on visiting an endemic area, are attacked almost immediately by the disease in its gravest form.

Nearly every inhabitant of the towns in these districts has acquired Verruga at an early age, and, if reinfected, manifests but the mildest symptoms.

There are two significant facts bearing on the etiology of Verruga. (1) One can become infected with the disease while travelling through the endemic zone, the contagion being most readily acquired at night-time, especially should one sleep in such a spot. (2) The disease occurs in newly-born infants, a circumstance which eliminates many channels of infection.

There is yet another fact of importance which, although not bearing the character of a scientific experiment, is nevertheless of great interest. Under the direction of Dr. Giaña the workers on a railway running through an endemic zone were obliged to quit the zone before sunset. Immediately the case incidence notably diminished.

Laboratory Investigations.—Up to the present time no one has succeeded in finding the specific cause of the infection. The ordinary laboratory animals cannot be inoculated with the disease. Although it has been said that some animals do contract Verruga, no one has yet definitely demonstrated that a certain disease with skin manifestations, found in places in Peru, is really the true Verruga as it occurs in man. A very superficial examination will not fail to convince the observer that this is so.

In the most severe cases without a skin eruption and ending fatally, Dr. Barton has isolated a paratyphoid bacillus, and latterly other bacilli belonging to the same group have been found by means of blood cultures during the patient's lifetime and in nearly every organ after death (Jamayo), more especially in the red bone-marrow. During the eruptive stage these baccili have not been found. The presence of this superimposed infection naturally materially affects the progress of the disease. Inoculations of cultures of these bacilli into guinea-pigs produce a fatal septicæmia.

Dr. Barton * has also found certain bodies in the erythrocytes (stained by Leishman) of Verruga patients which he considers may be protozoal in nature. He found them throughout the whole course of the disease. Drs. Gastraburú and Rebagliati have also found similar bodies in blood-films stained by Giemsa.

The bodies alluded to appear as very minute rods of a bacillary shape. They are variable in number and may stud the whole of an infected corpuscle. Generally they are thicker in the middle or at each end; sometimes, however, they are punctate. They stain a red-violet colour: at the same time they are very refractile. They have never been discovered outside the red cells. They are invariably present in every case and in every stage of the disease, more especially in severe cases. In these they are extremely numerous, occurring in almost every corpuscle. They become scantier after the eruption, and finally disappear during convalescence.

I have seen these intra-corpuscular elements in dry unstained preparations as little crystalline bodies, very refractile, and disposed in the manner described. Even in fresh blood I have sometimes succeeded in staining them by Widal, Abrami, and Brulés vital stain †. I found certain granular bodies in the red cells which resembled them in their general characters.

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* Gaceta de los hospitales, 1909, Lima.

† Journal Médicale, 1910 (Les ictéries hæmolitiques).

As to the exact nature of these appearances one cannot give an opinion at the present time. I have noted that these changes in the red blood cells are constant, and, moreover, that they exhibit a parallelism with the progress of the anæmia. Dr. Hercelles has found the malaria parasite in the blood of patients stricken with Verruga.

Symptomatology.—

(1) The incubation period of Verruga is nearly always about twenty days; in certain instances it is longer, possibly by several months. The first symptoms, which are found in every patient, are stiffness in the back, apathy, &c. These are soon followed by fever, anæmia, and severe general pains.

(2) *Anæmia.*—The rapidity with which anæmia is established (a feature found in no other disease) is a point of special interest. After three or four days, in the most severe types of the disease, the number of red cells falls to less than a million per cubic millimetre. Even in the mild cases the number of red cells is markedly below normal. Concurrently, all the signs accompanying pernicious anæmia, such as the presence of normoblasts, of which as many as 2000 per cubic millimetre may be found, sometimes megaloblasts up to 200 per cubic millimetre, together with every kind of alteration in shape and of degeneration in the substance of the red blood corpuscles. In the first stages of the disease a polymorphonuclear leucocytosis is the rule. At the same time the basophile myelocyte (not containing large granulations) makes its appearance in the blood. The presence of myelocytes containing large granules is exceptional. The colour index of the blood is raised.

The effect of this anæmia is seen in the earthy colour of the skin. The mucous surfaces are anæmic, the face is very pale, and hæmic bruits are commonly heard at the base of the heart.

The anæmia is so marked as to imprint upon the patient a facies extremely characteristic of the disease.

(3) *Fever.*—The temperature oscillates within wide limits and has no definite course. It is generally of a remittent type and hardly ever rises above 101° F. during the oscillations. Sometimes the characteristic eruption is seen in patients who have had no fever previously, but such cases are exceptional.

(4) *Pain.*—The pains are very marked and are the first symptoms complained of. Patients complain of horrible afflictions in the shape of pains in the head, joints, and bones. In others, abdominal pains are most marked, and are referred to the viscera, simulating in certain cases abscess of the liver. The bone pains are no doubt owing to the changes proceeding in the red marrow. The sensibility is so exquisite that pressure over certain bones causes great pain. This is specially marked and frequent over the sternum.

(5) *Other Symptoms.*—In the early stages there is often active delirium, and nearly always insomnia. An intractable diarrhœa makes its appearance, and often is the cause of an intolerable thirst. The liver is enlarged, but the spleen cannot be felt. In very thin subjects one can feel that the mesenteric glands are definitely enlarged.

The Urine.—The urine, during the feverish period, at times contains some albumen. Indican and urobilin are always present. There are no urinary hæmorrhages.

Severe Clinical Type.—Should the symptoms of the disease progress the patient sinks into a comatose state and dies. The duration of an attack of this sort is about ten days.

The Eruptive Form.—In other cases the symptomatology is less marked. There is a remission of all the severer symptoms; the fever falls, and with it the regeneration of the blood-elements commences.

The relative proportion of the various cell-elements changes, the eosinophile and mononuclears reappear, the number of red cells rises rapidly, the basophile myelocytes disappear, and the colour index returns to normal. To put it shortly, an examination of the blood at this time, always an indispensable proceeding in this disease and one of a great prognostic value, indicates a regeneration of the blood by the blood-forming organs. The pains decrease in intensity, though persisting till after the disappearance of the eruption. All the other symptoms are modified, and when the eruption makes its appearance the patient is practically convalescent.

Eruption.—At this juncture another phase commences, namely, the rash. This makes its appearance either when the septicæmic stage of the disease is not quite ended, or at a variable period after all the symptoms mentioned above have ceased.

Clinical Characters of the Eruption.—The eruption is seen in two principal forms, distinguishable by clinical characters, but similar in their pathological structure. These are called the miliary and the nodular eruptions.

Miliary Eruption.—This appears as little circular spots of a pink colour, and resembling after desquamation sudaminal vesicles. In a short time the spots increase in size and take on a bright scarlet colour, and their surface becomes smooth and polished. The patient suffers no inconvenience from their presence. The colour of these spots does not disappear on pressure. Immediately after this stage they become hemispherical in shape and may reach the size of a large pea, but they generally remain a little smaller. They preserve, however, their red colour, which merges into the deeper layers of the skin, though this on the whole retains a healthy appearance. Sometimes these little excrescences may become sessile. The nodule now becomes stationary. It is firm in consistency, and at first its surface is polished. Little by little it becomes changed into a dull red colour, shrivels up, and dries under an accumulation of crusts. When these crusts fall off the skin undergoes a slight desquamation, and there remains only a little stain which soon disappears without leaving a scar. This period of devolution, in certain cases, takes several months to complete. This is the natural termination of the eruption, though its character is often changed by the patients scratching, thereby causing small hæmorrhages or introducing secondary infections.

Nodular Eruption.—The nodular eruption, so called because the nodules are of a greater size, is felt, in the first instance, as a small and generally elongated body beneath the skin, where it can be rolled beneath the finger. On its first appearance the patient complains of slight pain. The lesions take a long time to develop; but when they have attained a certain size they elevate the skin, which is stretched over them, giving the appearance of a scarlet-red button. Gradually they grow larger, till they are the size of a haricot-bean, and even in some instances that of a lemon (eruption *mular*). These latter become pedunculated, strangulated, and gangrenous, and in this condition may necessitate removal by the surgeon. In many cases this eruption, though originating underneath the skin, becomes absorbed and disappears: in some instances, however, where they have grown to a considerable size, they gradually stretch the skin, which generally ruptures and hæmorrhages ensue. Being of a considerable size they are liable to secondary infections of all kinds. In other instances the evolution of these vascular tumours resembles that of the miliary form, in that they take more than a year to disappear, but being exposed to all kinds of external influences very easily become hæmorrhagic. This form of eruption is less common than the miliary form.

The Appearance of the Eruption.—Both the miliary and nodular eruptions may appear as one crop; more generally the eruption appears in successive crops at intervals of very variable duration. One can generally see certain nodules in the process of involution, while at the same time others are just making their appearance. In rare cases, after an interval of several months, and when the patient believes himself to be cured, another crop of nodules may appear.

Distribution of the Eruption.—Both forms of the Verruga are found on the skin; but the miliary eruption is so generalised as to make its appearance on the mucous surfaces as well as in the internal organs (the heart, iris, meninges, intestine, &c.), giving rise, in these situations, to all kinds of complications. On the other hand, the large nodular eruption is strictly confined to the skin, especially at the flexures (the elbows, knees, &c.), where it may form large tumours.

The first crop generally appears on the face and the extremities, such as the dorsal aspect of the upper arm, the forearm, and the hand, the antero-internal aspect of the thigh and leg. In some cases the eruption is discrete and is limited to a few nodules; but in others it spreads over the whole cutaneous surface of the regions mentioned and even on to the back and abdomen, and is then known as the confluent eruption. In certain cases no healthy area of skin can be found, two or three nodules endeavouring, so to speak, to make their appearance in the same spot. It is in these cases that an eruption is found simultaneously on the mucous surfaces. Drs. Biffi and Carvajal have reported a case in which the nodules underwent spontaneous suppuration without the intervention of any external agent.

The miliary eruption makes its appearance after the general symptoms have abated, but the nodular form is accompanied by fever (*Odriozola*). In cases, however, where the eruption is subintrant, the general symptoms recur before another crop of eruption appears. When the eruption is established a mononuclear leucocytosis (60 per cent.) is found and persists for a long time.

PATHOLOGICAL ANATOMY.

The Verruga.—The structure of Carrion's Verruga has been studied by Letuele, Jamayo, Escomel, Jeanselme, and Hercelles, and has been proved to be, both in the miliary and nodular form, of the nature of a neoplasm originating around capillary blood vessels, a neoplasm which is mainly formed of areolar tissue in response to a perivascular irritation. The connective tissue fibres are swollen, and between their interstices other connective tissue cells of an embryonic type are crowded. The interareolar spaces are swollen with plasma containing polymorphonuclear cells and macrophages. The blood capillaries are invariably dilated.

The Bone-Marrow.—The changes in the bone-marrow in severe cases, accompanied by pyrexia, have been studied by Carvallo [*], who found an increased production of normoblasts and neutrophile myelocytes in accordance with the leucocytic formula as found in the blood [†]. At the same time he found extensive lesions in other tissues of the bone-marrow, especially in the periarterial connective tissue.

Internal Organs.—Rarely in those cases of Carrion's disease which come to the post-mortem table does one find evidence of the internal eruption which I have found. Nicolle has also found in the internal organs miliary nodules, filled with caseous material, almost resembling miliary tuberculosis.

These are the most important facts which have been found so far.

Diagnosis.—An examination of the blood, demonstrating an anæmia of so pernicious a type, in a patient coming from a country in which the disease is prevalent, strongly suggests the diagnosis, a suspicion which is confirmed when the eruption has appeared. When the eruption is delayed the diagnosis is difficult.

Prognosis.—As to prognosis, this depends upon the enumeration of the red blood corpuscles. The appearance of eosinophile cells and of a mononuclear leucocytosis presages the crisis of the disease, and sometimes the appearance of the eruption.

On the other hand, an eruption of a very bright colour and an increase in the size of the spleen indicate that the disease will run a favourable course [‡] (*Odriozola*).

Treatment.—This is summed up in one word, there is none.

Pathogeny.—The cause of this disease is still unknown. The profound changes in the blood point to the blood-forming organs as being the original sites of the disease. The intervention of paratyphoid baccili, which have already been mentioned, may be considered under the category of those secondary infections which render the disease more dangerous. Nothing is known about the pathogeny of the disease.

I wish to thank Dr. P. H. Bahr for kindly translating this paper from the French original.

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[*] Thèse de Lima, 1911.

[†] Monge: Thèse de Lima, 1911.

[‡] It the reader desires more complete information on the disease he should consult the splendid monograph of Dr. Odriozola, which gives all that is known about Carrion's disease up to the present (Gaceta de los hospitales, Lima, 1910).

REPORT FOR THE YEAR 1910 OF THE AUSTRALIAN INSTITUTE OF TROPICAL MEDICINE.

By A. BREINL, M.D.,
Director of the Institute.

In January 1910 Dr. Breinl arrived in Townsville, Queensland : the work of organising a laboratory had then to be undertaken. A not very adequate building appears to have been practically all that was ready for him. Great difficulties were experienced also in procuring suitable laboratory animals for experimental work in Townsville. Efforts to breed rabbits failed, and of large batches imported from New South Wales nearly all died. Only five monkeys were obtainable ; tame mice could not be had ; wild rats, easily captured, did not live long in captivity ; guinea-pigs were only procurable from the South in small numbers. Under present conditions it is, therefore, impossible to carry out experimental work on a large scale. Dr. Breinl, in spite of these difficulties, has been able, however, to make a contribution of great interest to Tropical Medicine concerning the geographical distribution of disease. Queensland is a large country, though only a part of Tropical Australia, and to-day possesses but a scanty and scattered white population. There are no villages with a dense native population. Cases of disease occur, only in few numbers, distributed over a wide area. Reviewing these conditions, Dr. Breinl draws attention to the fact that while they make sweeping epidemics impossible, they also render more difficult the study of the local diseases and their distribution. Nevertheless, it is of great value to Queensland to have such an Institute of Tropical Medicine already started. The diseases and their distribution noted now will form the standard with which can be compared, in future times, the prevalence and spread of these same diseases or any new ones that may be introduced. The difficulty of preventing, under present conditions, introduction of disease is dwelt on. Many tropical ailments cannot be diagnosed clinically at a first examination of the patient. The Institute, however, will serve the purpose of watching for the advent and spread of new diseases, and in the meantime of establishing the best prophylaxis possible, by training Australian medical men in Tropical Medicine. Queensland is enjoying at present, on account of its isolation and scanty population, a comparative freedom from epidemic diseases—a freedom, however, which can only be temporary. Population is increasing and will increase more rapidly as time goes on ; its over-sea commerce is extending, bringing it into more intimate contact with the peoples dwelling in the great tropical archipelago between Australia and Asia. Also railway communication with the South and between the Northern coastal towns of Queensland will provide a means for the introduction and spread of various deadly diseases which exist in the near neighbourhood. The incubation period of some of these diseases is prolonged and the diagnosis, especially in the earlier stages, difficult and often impossible. Even strict quarantine regulations are, therefore, not to be depended on, and it will prove a very difficult task to keep these diseases out for ever, even if there were appointed a highly scientifically trained staff of quarantine officers.

The question of a white race living and rearing strong and healthy children in the Tropics is to-day declared to be only a question of sanitation. At present in Queensland the eradication, at comparatively small cost, of the existing diseases should not be difficult. It is not an impossible task to lessen the danger of introduction and prevent the spread of new diseases. To accomplish this, however, the health officers must possess a knowledge of all the epidemiological factors of the threatening diseases, only to be obtained by intimate acquaintance with their clinical and parasitological phenomena—especially as some diseases can be introduced by intermediary hosts such as ticks, mosquitoes, biting flies, &c. It is necessary, therefore, to possess an exact knowledge both of local conditions and the presence of possible intermediary hosts. The Institute has before it the great task of pointing out how Tropical Australia shall be made "a white man's land."

The study of this question and the results obtained will prove of incalculable value to other peoples outside Australia. The Institute has, literally, a wide field for research—all Tropical Australia and the neighbouring islands as far as Papua. In this report Dr. Breinl gives a concise account of the prevalent diseases of a tropical nature met with in Queensland and the adjacent islands, Papua, Torres Straits Islands, Thursday Island, &c.

They are Sprue, Amœbic Dysentery, Filariasis, Malaria, Beri-beri, Ulcerative granuloma, Yaws, Dengue Fever, Leprosy and "Sandworm" disease, and "Barcoo rot." Of these diseases only a few cases were met with as a rule: malaria, dysentery, yaws, beri-beri, and leprosy being chiefly confined to the islands to the north of Queensland.

Malaria is known to be endemic in Queensland in certain districts—Innisfail, Alice River (near Cooktown), and the Saxby River. It is mostly of the benign tertian type. Crescents were found in the blood of persons from the Fly River in Papua. The traffic between N. Queensland and Papua being frequent, there is a danger of malignant malaria reaching the mainland from neighbouring islands.

Dysentery occurs at times in epidemic form in Papua and Thursday Island. In Queensland itself dysentery is seldom seen—Dr. Breinl's only case was amœbic in nature.

Beri-beri of a mild type was seen in Thursday Island.

Dengue fever in 1910 was very common in Townsville; careful search did not reveal any specific organism in blood or secretions.

Ulcerative granuloma.—Three cases were studied by Dr. Breinl—one a white man, two aboriginals. In one case large numbers of *Leishmania*-like bodies (as described by Manson) were seen. No spirochætes were found.

Filariasis.—A common disease in Queensland. Orchitis, hydrocele, varicose groin glands, elephantiasis, chyluria and hæmato-chyluria, and filarial lymphangitis are all frequently met with.

Leprosy.—Mostly seen in Thursday Island: the report contains interesting details.

Intestinal parasites found to exist in Queensland and the Islands are:—*Trichocephalus dispar*, *Oxyuris vermicularis*, *Ankylostoma duodenale*, *Strongyloides stercoralis*, *Tænia saginata*.

Ankylostomiasis is the most serious of these infections. It is widely spread, among school children and adults alike. It is of general significance and calls for treatment in radical fashion if its extermination is to be brought about. Dr. Breinl notes a lesion which apparently precedes the ankylostome infection — especially in the Innisfail and Cairns districts. The lesion generally occurs on the inner side of the sole of the foot. First a small inflammatory area appears, soon spreading in the form of spirals and disappearing after a shorter or longer period. Careful work is needed before deciding whether this peculiar lesion is to be compared with the "ground itch" of Assam, or the "Craw Craw" of the West Coast of Africa. It is a popular belief in Cairns that the lesion is produced by the entrance of a "sandworm" under the skin. Experiments on monkeys were therefore carried out with "the sandworms" but proved negative.

"*Sandworm*" *disease.*—A peculiar inflammation of the skin occurring mostly on the inner side of the foot. Its etiology and pathology at present are not clearly understood. One case was carefully examined by Dr. Breinl, dissecting as far as possible into the flesh, but failing to find anything which might give an explanation of this curious lesion.

Barcoo rot.—A skin-disease occurring in Western Queensland. Small, round, superficial ulcers with irregular outlines: the ulcerated surface soon dries and becomes covered by a scab; under this scab healing takes place, but ulcers break out again on other parts of the body, especially on legs and arms. Bacteriological examination of one case did not yield any results. Unsuccessful attempts were made to transmit the disease to the shaved skin of a young rabbit.

Dr. Breinl gives an interesting series of photographs and descriptions of a peculiar chronic ulceration of the Nasopharynx, which closely resembles the condition known in Fiji as "Kaninloma," but found also in S. America and Africa, and by many suspected of being a tertiary manifestation of Yaws. The same disease was described as a separate disease in the Island of Guam as Gangosa or Granuloma Gangrenose. Dr. Breinl notes the special frequency of the disease in Murray Island and considers it to be undoubtedly contagious.

Considering the great difficulties Dr. Breinl had to contend with in starting work and the vast extent of country he has had to survey pathologically, he is to be congratulated on the work already done ; and this, the first report from the Institute of which he is Director, bears promise of future achievements in Tropical Medicine of no less value to science than those already gained by Dr. Breinl in Brazil and Africa, and at the Research Laboratories of the Liverpool School of Tropical Medicine. H. M. H.

FOURTH REPORT OF THE WELLCOME TROPICAL RESEARCH LABORATORIES.

GORDON MEMORIAL COLLEGE, KHARTOUM.

Fallacies and Puzzles in Blood Examination.

(Being an account of the more common deceptive appearances met with in such work, especially in tropical countries.)

By THE DIRECTOR.

In an introduction to his well-illustrated paper Dr. Balfour says :—"It has occurred to me, for those at least who are novices in the art of blood examination, and more especially for such resident in tropical countries, a short paper on some of the more common sources of error encountered in this work may be useful. It is true that some of them are mentioned in the text-books, more especially in volumes devoted to malaria ; but even in these I find there are many omissions, and one has to search through papers on various subjects before one gets an idea of all the pitfalls into which the hæmatologist may be plunged. Moreover, it would seem that there is an urgent need, in English at least, for some such article. From time to time one finds descriptions of so-called blood-parasites, either in man or animals which it does not require a Solon to diagnose as being in reality one or other of the conditions we propose to describe, and not hæmatozoa at all."

Dr. Balfour cites two recent instances of papers on blackwater fever. In one were described piroplasmata in stained films and free forms in fresh blood, and the suggestion made that this "piroplasm" is the cause of blackwater fever. Balfour believes the author had confused several blood-conditions, one doubtless pathological, the other histological. In the other "spore-like" forms were stated to be found in both fresh and stained specimens of blood. There is little doubt but that these "spore-like" forms were derivations of the blood itself, though the author of this second paper was possibly on surer ground when mentioning the curious X-bodies of Howell and Horrocks. It is a pity if time be wasted and other workers possibly misled.

The paper continues:—"One cannot hope to deal with every source of fallacy which may occur any more than one can hope to describe every type of puzzling and peculiar condition, but by grouping together and *illustrating* in one paper the more frequent sources of error and difficulty, it may be possible to help those who are unfavourably situated for the obtaining of advice either from books or from more experienced observers."

For convenience, one may divide these conditions into two classes according to their origin :—

I. External, Adventitious or Heterogenetic.
II. Internal, or Autogenetic.

Under I. come cases in which the source of fallacy is derived from outside the blood itself, as, a classical example, the inclusion in a film of insect hairs simulating filariæ. Low and Stiles take this view of the *Filaria gigas.* Again, cotton-fibres gaining access to blood-films led to a wholesale diagnosis of filaria cases. This type of fallacy, care and cleanliness, the watch-words of a hæmatologist, should avert.

Class I. is more likely to be found in stained blood—an examination of the fresh blood-film often clears up a difficulty. Yeast-cells frequently occur in films made in the Tropics. Chromatin-like granules in such cells, definite arrangement of a capsule or enclosing membrane, frequent presence of definite segments, form objects apt to be confusing. Such bodies are not likely to gain access to fresh-blood preparations, which have perforce to be made quickly. Possibly also yeast-spores may develop to some extent on blood-films and produce confusing pictures. In the same category, though not so common or so puzzling, come pollen-grains and animal and vegetable débris from dust. Attention is drawn to the possibility of intestinal parasites getting into blood-films. In this connection, Balfour quotes from a paper of his on the finding by Nuttall of a new spirochæte in the African buffalo and gives Nuttall's reply. Wenyon has seen forms like *Spirochæta bovis caffris* (Nuttall) in the blood of big game which had been shot; and recorded his opinion that the forms in question were not blood-parasites at all, but were derived from the intestine, being carried into the exit-wound by the bullet or by discharges finding their way along the bullet-track. The possibility of such an occurrence and the fallacies to which it might give rise are noted, and insistence is made on the recognition of this source of error while the question still remains open.

Nuttall's drawings of his spirochæte are reproduced side by side with illustrations of very similar bodies found by Balfour in blood-films from a Jackson hartebeeste, shot through the neck, severing the gullet; it being quite possible that, in the last throes, stomach-contents might have regurgitated through the wound of exit. The finding of thin thread-like spirochæte forms and yeast-like bodies in the film enjoined delay in publishing an account until more evidence regarding Wenyon's hypothesis had been obtained. Recently he had received from Capt. Cummins, S.M.O., Kordofan, a blood-smear from a goat which had died from infectious pleuro-pneumonia, Capt. Cummins having found spirochæte forms in the film, which Dr. Balfour recognised as his hartebeeste parasite or forms very like them. On further enquiry, Capt. Cummins stated there had been every possibility of contamination with contents of intestines, these having been examined just before making the blood-film; and agreed that the parasites were of intestinal origin. Professor Nuttall in his reply states: his films were contaminated only with a few bacilli and were made from blood obtained, as it welled up, from a stab in the heart of a buffalo (No. 1). The hunter was "sure that there was practically no chance whatever of organisms from the intestines getting into the blood circulation." After the films were taken contamination was possible; accelerated manipulation being necessary, another buffalo (No. 2) showing a desire to charge the hunter. No. 2's blood contained no spirochætes. The nature of the organisms can only be decided by further observations: they differ considerably from the typical spirochætes which various observers have found in the intestine.

Parasite-like bodies from the skin and hair of animals may appear in films. A flagellated form found in a blood-film made from a guinea-fowl is illustrated. It cannot be said exactly what it is, but a shrewd suspicion is expressed that it was derived from the feathers. Reference is made to the peritrichous ciliate, probably a cyclochæta (Ashworth) from the gills, found by Neave in the blood of a fish (figured in Second Report, 1906). Pigmented bodies found by Major Howard Ensor in human blood-films are here illustrated. They appear to be artifacts and possibly have an animal origin. In a case like this, a careful search of the film helps to a decision if not to a diagnosis, for it is rare to find only one example of a true blood-parasite in any given film. This may occur, especially as regards malarial crescents and filariæ, but reflection and doubt are recommended if in, say, two good-sized well-made blood-films one can only discover a solitary example of what seemed at first a new and peculiar hæmatozoon. It is well also to follow Daniels' advice, and to examine some part of a slide where the stain has extended beyond the blood-film and see if the same appearances are there. It is worth noting that in Australia birds' (Honey-eaters) spermatozoa, the long and narrow types of which show regular spiral curves, were mistaken for spirochætes. The spermatozoa had passed into the blood-clot owing to the shot-wound injuring the spermatic duct. Returning to yeast-cells, it is found they present very varying forms. In this Report are shown illustrations of what is believed to be a form of yeast-cell which was associated with a cutaneous Leishmaniosis, and led more than one astray. A torula from the stomach of a jerboa (figured in the Second Report) under certain conditions might quite possibly deceive the very elect.

The amount of literature accumulated on the parasite of epizootic lymphangitis shows one effect of what may be called the yeast-trap. Able observers have considered it a Leishmania or leucocytozoon—it has been definitely shown to be a yeast, and has been cultivated as a fungus growth on artificial media.

The points to consider in distinguishing a stained yeast-cell from a blood-parasite are: the history of the film: the general shape and appearance of the yeasts. The portions of yeast cytoplasm taking on the chromatin dye stain a very intense ruby-red; they are also, as a rule, more compact and "solid"-looking than nucleus or blepharoplast of a hæmatozoon. The appearance of the capsule helps, *e.g.*, the double contour of the parasite of epizootic lymphangitis. In some cases, however, the only thing to be done is to make a careful drawing and record the find with such particulars as may help, mentioning especially any other forms—bacilli, cocci, &c.—which may indicate contamination. (A coloured illustration is given of a blood-film from a Uganda cob showing contamination with yeast-cells and other curious bodies simulating parasites.)

Experience and careful reasoning will usually prevent mistakes in Class I. There is another and most important fallacy. It is a common custom to dry a stained blood-film, after washing, by means of blotting-paper. The piece of filter-paper, the under surface of which is pressed down upon the film, must be of virgin purity. Films of mammalian blood showing hæmogregarine infection, prepared for transmission to India, were looked over before despatch. They were found to contain oval and nucleated red cells, which must have come from bird or reptile. It was concluded that either the slides, the pipettes, or the spreading-needle used had been dirty. A similar episode occurring again, it was realised that the same piece of blotting-paper used to dry a stained film of fowl's blood had been used to blot the other film and the corpuscles had been transferred from the paper to the slide. Dr. Balfour knows of cases in which parasites were transferred in a similar way. Fragments of stained epithelium and stain deposits often spread beyond the margin of any red cell on which they may lie, and may be troublesome, though focussing helps to decide. Finally, skin-pigment or dirt must not be called melanin, which never occurs free: cocci and bacteria get into blood, and there are sometimes strange flaws and cracks in glass which may retain a stain.

Class II. Autogenetic fallacies are considered under two headings: (1) Those found in fresh blood; (2) Those present in stained preparations.

(1) Crenations of the red cells have no defined outline, and change from dark to light or reversely on altering the focus, being thus readily distinguished from a malaria ring form, piroplasm, or spirochætal granule or body. Malarial crescents contain pigment, distorted red cells do not. Little protoplasmic masses in the blood containing dancing pigment, with perhaps a ruptured red cell near by, are the severed rolled-up amœboid processes of a half-grown benign tertian parasite. Vacuoles must be distinguished from a young malarial parasite, a piroplasm, or a spirochætal inclusion, this last being especially difficult. The following are the points to be considered:—

(i.) Vacuoles have well-defined clear-cut margins, while the periphery of a parasite melts, as it were, into the hæmoglobin of the host cell.

(ii.) A vacuole may change slightly in shape—this must not be overlooked,—but it never shows true amœboid movement.

(iii.) A vacuole never shows pigment nor any appearance suggestive of granules in its interior or projections from the vacuoloid area.

(iv.) A vacuole is clear, with no "ground-glass" aspect; it is highly refractive and on focussing up and down seems to spread and contract in a characteristic fashion.

(v.) Vacuoles never stain as a whole, though there may be deposits of colour round their margins.

Cracks or splits in red cells are easily recognised, but among the most difficult of all blood-appearances are those furnished by areas devoid of hæmoglobin—called "eye-spots," "hyaline bodies," &c., "Maragliano's endoglobular degeneration" or shortly "Maraglianos." These are found in normal blood, especially in that of small rats and gerbils, but are more abundant in certain pathological conditions, and are very apt to be mistaken for hæmatozoa. They tend to occur in acute infectious conditions, are found in any part of the red cells, vary in shape, and sometimes appear to have some kind of structure. They are not clear like vacuoles; and, especially with dark-field illumination, the appearance of a tiny waving filament projecting from their centre has been observed. They have no amœboid movement, but seem capable of changing their position in the red cells, without any pressure on the cover-slip or disturbance of the blood-film. The movement appears due to progressing coagulation and consequent constriction of the protoplasm (Webster). They are round, oval, and spindle-shaped, and may be multiple. The elongated forms often have a little dark spot, so distinct as to look like pigment. They usually change a little in size on focussing—some help in distinguishing them.

Cabot's nuclear remnants, occurring in pernicious anæmia, leukæmia, and lead-poisoning, as also Cropper's bodies, met with in cases of fever, are probably of this class.

Reference is made to the plates in Buchanan's 'The Blood in Health and Disease' (1909) as being helpful to those to whom abnormal red cells present difficulties; the book is very useful to anyone studying blood morphology and more especially cellular degenerations.

Granules from broken-down leucocytes may overlie red cells. They have no amœboid movement, no ground-glass appearance, vary greatly in size, and are differentiated easily by focussing. If a doubtful body is seen lying apparently in a red cell, the plasma should be searched to see if its exact counterpart is not to be found lying free. The red cells may undergo curious degeneration, especially when the temperature is raised. These changes have been described and illustrated by Nuttall and Graham Smith. These alterations in shape may be called "the chain, dumb-bell, droplet, and filament phenomena," a comprehensive and descriptive, if clumsy, term.

Even in the height of a Sudan summer these changes were never encountered in blood-films examined at room temperature, at least when using transmitted light, though they are at first very puzzling when working with heated blood. Nuttall and Graham Smith pointed out that these changes may occur if the platinum loop used to transfer a drop of blood to a slide is not cooled sufficiently, though the glow may have faded, after sterilisation. It may occur also if a finely drawn glass pipette be used immediately after it has been made. In fresh blood, a platelet lying on a red corpuscle may simulate malarial plasmodium; it is, however, less opaque, has a granular look, and is less refractive—often it presses the haemoglobin of the red cell away from under it and so looks as if surrounded by a pale area. In the case of any doubtful body lying on, or apparently in, a red cell, help will be obtained by altering illumination of field. A dull light is responsible for many errors, and brighter illumination may change the colour of a dark object from black to bright crimson. Touch the edge of a cover-glass with a needle and note if the body moves with or independently of the red cell. In the blood of cattle especially, platelets may take on flagellate-like shapes—that these thrombocytes always tend to unite in clusters helps in distinguishing them. A mass of platelets in human blood, surrounding a little pigment-like dirt, may look like a sporulating malarial parasite; but platelets have ragged edges: recourse can be had to staining, while blood examination should never be disassociated from clinical conditions. It is noted that the latest work on the histogenesis of blood-platelets goes to show that they are detached portions of the cytoplasm of the giant cells of blood-forming organs (Wright). Swingle has shown that blood-platelets in blood ingested by an insect host may closely simulate herpetomonadine flagellates. Dr. Balfour thinks errors may arise in fresh blood if this source of fallacy is not borne in mind.

Blood-dust, or hæmoconia, does not simulate parasites; these little dancing particles can only prove a stumbling-block for those who seek to diagnose malaria from the presence of free spores in fresh blood—a proceeding emphatically condemned by Deaderick. They may be confused with granular leucocytic débris—a matter of no moment,—with micrococci or fat-droplets. This last not an error, for recent work of Neumann and others goes to show that blood-dust consists really of fat-particles possessing Brownian movement. They are more numerous after a meal, and disappear after starving. They may be absent in disorder of the alimentary tract.

Some of the appearances last described, more especially blood-dust and droplet degeneration of red cells, are much better seen with dark-field illumination. Dr. Balfour believes the day will soon come when this method will be employed as a routine procedure and as a method of differential diagnosis, and gives a most interesting account of the appearance of microscopic fields observed by the dark-field illumination. The first thing to strike one is the enormous number of minute dancing particles, varying in size from 2 μ diameter to the tiniest specks, which, being brilliantly illuminated, look like infinitely minute sparkles. The larger particles vary in shape, being round, oval, rod or dumb-bell shaped : and while the smaller are solid the larger may appear vesicular. As noted by Porter, in these, which are highly fractile, can sometimes be seen a central spot of a ruby colour. Crawley has described particles exactly like blood-dust, which, from focussing tests and their limited movements, apparently were intracorpuscular, and suggested that some of the blood-dust must therefore consist of broken up erythrocytes or fragments derived from them. These had never been observed, but fat-particles by osmosis might find their way into red cells. In ordinary fresh blood-films, viewed by transmitted light, spherical bodies have been seen which certainly suggested oil-globules. According to Love, hæmoconia are especially numerous in blood of typhus-fever patients. Crawley has recently described "beaded threads," changes similar to the chain dumb-bell droplet and filament phenomena exhibited by the red cells under influence of heat. They can be seen by the dark-field method when there is no possibility of the blood temperature having been raised—at least appreciably so. Crawley also noted free flagella. These are probably of the same nature as the beaded threads and probably the same as "filaments"; but it must be remembered that, under artificial conditions, lymphocytes (Ross and Macalister) and myelocytes (Buchanan) may flagellate—induced by certain excitants, atropine and methylene-blue, as well as by a cancerous plasma. It is possible that certain pathological conditions of the blood may bring about flagellation in white cells, and that free flagella may account for some errors of diagnosis. Crawley notes its occurrence. Under dark-field illumination, bodies with pseudopodia have also been seen in normal blood—clearing up a fallacy, Koch and Kleine at one time supposing them to be associated with piroplasmosis in dogs and cattle. They are only greatly altered blood-cells, more probably derived (Crawley) from erythrocytes than from leucocytes.

As noted by Crawley, the heat of the dark-field method has a stimulating effect on the white cells. Balfour found bodies in red cells in the fresh blood of fowls, and supposed them to be a special form of intracorpuscular spirochætal inclusion—recognised to be an error by finding similar bodies in blood of gerbils, which, though inoculated with spirochætal blood or blood supposed to contain spirochætes, never showed any sign of illness. These bodies are held to be analogous to the intracorpuscular vesicles seen under dark-field illumination by Crawley ; they have been seen to grow smaller and become the nebulous clouds of which he speaks. True and pseudo-intracorpuscular forms look exactly alike. Crawley states that the pseudo forms are motile, a point which may serve to distinguish them.

Blood-plates are better studied by transmitted light ; it is said that the dark-field method has shown that mononuclear leucocytes break up into bodies like hæmatoblasts. If confirmed, this observation, while not of much importance in the examination of fresh-blood specimens, may well explain some puzzling features met with in stained films of animals' and birds' blood.

Eve's paper on leucocytic degeneration in the cerebro-spinal fluid is suggestive. The illustrations he gives show that if similar changes can occur in the blood, and under certain conditions nothing is more likely, then there is every probability of the resulting motile, globular, hyaline, and flagellated bodies which are produced being mistaken, as they were at first by him, for extraneous protozoal contaminations.

Passing now to consider the fallacies and puzzles, of an autogenetic nature only, in stained blood. Pride of place may be given to the little blood-platelets, the commonest source of error, in the blood. A blood-plate resting on a red cell may, as in fresh blood, lead the inexpert to diagnose malaria. It is distinguished by having no blue-staining protoplasm; its chromatin is deep red, irregular and granular: its outline, as a rule, indefinite, and generally it has pushed the hæmoglobin away from under it. A mass of platelets has been mistaken for a crescent or a clump of spores—their staining and the lack of pigment should leave no room for doubt.

Platelets sometimes show dark chromatin in rods very like blepharoplasts: careful observation and the considerations already mentioned will differentiate them from Leishmania. The surest guide to their identity is their reticular structure. Osmic-acid fixation probably preserves their form better than any other method. In animal blood the platelets may assume confusing forms: flagellated and blue-staining forms were illustrated in the Second Report. A red cell is often seen apparently extruding platelets—a phenomenon never apparent (H. Ross) when blood is examined by *in vitro* methods. He has drawn attention to the diffusion vacuoles in platelets; these appear as red spots when stained. In fowls the so-called thrombocytes take the place of platelets. As they are large cells, staining quite deeply by Romanowsky method, they may be mistaken for hæmogregarines (illustrated in Third Report)—Von Recklinghausen named them "*Spindelzellen.*" They are most likely to be mistaken for distorted lymphocytes. Burnett describes them as elliptical, oblong, or spindle-shaped, with an elliptical to broadly oval nucleus. The cell has nearly the length and about one-half the breadth of the average red cell. The nucleus, usually in the centre, occupies one-half the length and nearly the entire width of the thrombocyte. The cell-body is pale and often contains one or more clear vacuoles and occasionally one or more compact, rounded, deeply staining (deep purple, Wright's stain) bodies, about the size of, or somewhat larger than, a mast-cell granule. These bodies are probably a result of degeneration. Thrombocytes show a marked tendency to collect in clumps. In fresh blood and in the thicker parts of films they collect in masses in which it is difficult to distinguish the outline of individual cells. This indistinctness of outline and structure show their vulnerability. When taken from the blood-vessels they quickly pass through a characteristic series of changes. Cell-body and nucleus become less distinct, the cell-body losing its structure first. Finally, both become structureless, appearing in stained preparations merely as a diffuse stained mass, the nucleus being distinguishable by having a slightly deeper stain.

Alterations in shape and size of red cells are well known and need not be dwelt on. Illustrations are given of puzzling forms of distorted and enucleated erythrocytes seen in the blood of the fowl—probably caused in spreading the film. So-called hæmoglobin drops are met with in birds' blood: they look like human red cells. Coloured figures are given of the *corps en pessaire* and *corps en anneau* of the French authors, seen in blood of malarial cachectics, as also of fragile red cells, assuming half-moon and other peculiar shapes, met with in chronic malaria. Endoglobular and anæmic degeneration of red cells with a deposit of blue stain at their centres may simulate a hæmatozoon, but the absence of chromatin and the general distribution of the stain are distinctive. There is more reason for mistake in dealing with granular or bacillary-like basophilia, especially when associated with polychromasia. Basophilia is very common in blood of all kinds of rats. Indeed, it is held by some that certain so-called basophilias are not histological changes at all, but are due to parasite invasion. Graham Smith (1905) described bacillary-like bodies with protozoal staining reactions in the red cells of the mole. These were like the red forms found by Balfour in the gerboa and declared merely to represent basophilia by Laveran. They do not usually stain blue with Romanowsky, and in cases of the kind the tinctorial reactions are of the greatest service in arriving at a diagnosis. They may, however, do so. There are also different kinds of granules.

Naegeli's azurephil granules, probably due to degenerative changes in old erythrocytes, are quite distinct from what Ferrata calls true blue basophilia, which have a regenerative significance and consist of what Pappenheim calls parachromatin, a substance allied to the chromatin of which the Jolly bodies are composed.

Cabot's ring-bodies are rare; they occur in acute, large lymphocyte leukæmia and pernicious anæmia. They stain red both with Romanowsky and with methyl-green-pyronin. Sometimes true basophile granules may be very large like those met with in embryonal blood. Recent work on staining the living blood has led to the discovery of the granular-filamentous substance of Cesaris-Demel and his metachromatic-granular material; however, in ordinary blood work, consideration of these can be discarded. Pappenheim places in a special class the Maurer and Schüffner dot-phenomena of malaria.

In a film taken from a gerbil which had been inoculated with finger-blood from a relapse case of human spirochætosis, but which never showed infection, an appearance was noted in a polychromatophilic red cell as of a chromatin streak ending in a short flagellum. Possibly it is due to a stain deposit in a corpuscular split or crack. It is probably allied to the ring and figure-of-eight bodies (*corps en anneau*) mentioned by Schleif, Webster, Brumpt, and others, or to the intracorpuscular polychromatophilic filaments known to occur in red cells.

Very often in blood of rodents, especially rats and guinea-pigs, stained by Leishman or Giemsa methods, are found small spherical chromatin dots in the red cells, recalling in all but position *Anaplasma marginale* of Theiler, now said to be the parasite of gall-sickness in cattle in South Africa. These little red bodies first described by Jolly bear his name alone or are called Howell-Jolly bodies. Ferrata has studied them. Most commonly they are single, but two may occur, one often larger than the other; and they may be combined with punctate basophilia. It is important to note that they have been seen in human blood in cases of leukæmia. Study of the normal bone-marrow apparently showed them to be chromatin residues from enucleated erythrocytes; but Jolly recently put forward the view that they were due to hydration of the stroma or corpuscular membrane, and believes that polychromatophilia, granular red cells (seen in unfixed films), and basophilic granulations are only different degrees of the same change.

Balfour here points out that a study of embryonal blood clears up many obscure points in blood-cell morphology. These nuclear remains, if such they be. stain red with Leishman and Giemsa, and green with methyl-green-pyronin. A very puzzling conjunction, frequently overlooked, is a nuclear fragment situated in a basophilic red cell, which stains blue like a leucocyte fragment.

Theiler's marginal dots, in cases of piroplasmosis in cattle, had been until quite recently considered by most to be of this nature. Now, however, he and Spreull believe them to be parasitic in nature, and he has named the new protozoon *Anaplasma marginale,* and apparently definitely proved it to be the cause of gall-sickness. They may occur free in the plasma (Spreull). Whether or not this work is confirmed, it behoves all workers in the Tropics not lightly to dismiss chromatin-staining bodies in red cells as nuclear remnants, but to find out if they have any clinical and pathological significance, to look for them in the fresh blood, to test effects on them of different methods of staining, and to carry out suitable inoculation experiments.

More especially is this true if they appear to have a selective affinity for any special portion—*i. e.*, the margin—of the host cell. Just as the free nuclei of normoblasts in a film are explained by the presence of nucleated reds, so difficulty from megablasts showing polychromatophilia, especially puzzling if their nuclei are undergoing degeneration, is cleared up by finding ordinary megablasts in the film. In the red cells of birds may be encountered the extra-nuclear centrosomes of Ross, Moore, and Walker. They have also described them in human corpuscles. They stain bright red with polychrome-methylene blue and occur as single granules, often connected by filaments to other granules, forming a group. The result is a kind of star rosette. They occur along with the chromolinin granulations of these authors, and may be shown by their methods of vital staining. These are the granular thread-forming (Fäden formige) substance recently described by Ferrata and Boselli.

Turning now to the white cells for the fallacies and puzzles they may yield in stained blood preparations. Leucocytes of pathological significance are not considered. Myelocytes, different kinds of basophiles, mast-cells, &c. can usually be recognised without much difficulty. One has rather to deal with fragments of leucocytes, inclusions in them, curious nuclear changes and conditions which may suggest blood-parasites either in leucocytes or free in the plasma and yet derived from them. For instance, the spherical homogeneous blue-staining bodies, so often met with in spleen-smears in kala azar, may be found in the peripheral blood. They are quite distinctive and are, in all probability, fragments of host cells (macrophages), which having been broken off assume the spherical shape in the circulating blood. This, however, is by no means proved.

Attention is directed to curious forms (portrayed in the paper) from the blood of a donkey, possibly a recovered case of piroplasmosis, and from the blood of a gerbil which had been inoculated with negative results from a goat, which in its turn had been inoculated with the camel trypanosome of the Sudan. One form is not unlike the bodies into which mononuclear leucocytes may break up, and it must be remembered that in severe and advanced cases of piroplasmosis (equine, canine, or bovine) the blood may show marked degenerative changes and present curious pictures of cellular vagaries. From the same donkey's blood is pictured what is almost certainly merely a degenerated leucocyte where the nucleus has undergone chromolysis. Possibly in the same category come further curious cells portrayed, from the blood of a fowl dying in the spirochætal "after phase" or "granule phase." Myelocytes were present in its blood and every evidence of great blood destruction.

Some of these appearances may be understood in the light of Ross' work on achromasia of the leucocytes and the changes which occur when they die. After their death polymorphonuclears and eosinophiles often appear mononucleated—possibly due to liquefaction of nucleus, its lobes running together. Liquefaction of the cytoplasm is said to begin at the periphery and progress towards the nucleus; while in achromasia the granules of leucocytes turn red. It is well to keep in mind that in blood-films we may find white cells which had been circulating as dead matter, and therefore exhibit changes which are never seen in living cells. Apart from actual death, degeneration and vacuolisation may make leucocytes almost unrecognisable, especially if there is much karyolysis of their nuclei. The clinical condition, the history of the case, other blood-findings, and the evidence of grave anæmia will usually lead one to a diagnosis of such abnormal cells. They cannot well be mistaken for anything else, and their outlines are too broken and irregular for them to suggest protozoa. A picture is given of a parasite-like body—ring-like and with well-marked chromatin body—from blood of gerbil inoculated without result with human spirochætal blood. It is perhaps derived from a white cell. Oval or round rings may occur in the cytoplasm of lymphocytes. Certain forms—centrosomes (Ross)—occur when blood is kept on a jelly and stained with an alkaline stain. They lie above or below the nucleus, not in it. It is possible that such rings may show, under certain conditions, in the peripheral blood. Balfour has seen in the extra-nuclear portion of a camel's mononuclears tiny chromatin-like and very perfect rings, the significance of which is quite unknown to him. They were not numerous, and he does not think they had anything to do with the trypanosomiasis from which the animal was suffering.

"Ruby" granules found lying free in a blood-film stained by Leishman's method are almost certainly derived from a lymphocyte; they are not common, for the lymphocyte is not a very friable cell. They stain quite differently from free eosinophile-granules. Recent blood investigations have shown the frequent presence of special granules or bodies in the extra-nuclear cytoplasm of certain of the mononuclear elements. The most important of these are known as Ferrata plasmosomes and Kurloff's bodies. The former are small, found in all types of lymphoid mononuclears staining metachromatically, and by Ferrata are thought to be evidence of regressive metamorphosis. The latter, larger (specially studied by Schilling), have been considered to be secretion vacuoles or—important from the point of view of this paper—parasites. It has been suggested that the intracorpuscular stage of fowl spirochætes described by Balfour in red cells was in reality of the nature of plasmosomes or Kurloff's bodies. It is important to bear them in mind. Schilling's work, from observations made on the blood-forming organs and tissues of the guinea-pig, goes to show that they are merely phagocyted structures (the action having taken place during blood-formation), which undergo only regressive change after absorption by the mononuclear cell.

Kasarinoff's work on the blood of pigeons ('Folia Hæmatologica,' 1910) with his plates of normal and abnormal cells and curious blood-findings are well worthy of close attention. Of special interest are the polar bodies he describes in thrombocytes after treating the blood with saponin. They appear as small single dots, spherical solid bodies, or as rings with solid inclusions, as signet shapes or as little spore-like clumps. The nature of these has not been settled. Save for their azurephil staining reaction and the fact that they occur in thrombocytes, they closely resemble some of the bodies in fowls' red cells believed by Balfour to be spirochætal inclusions. It may be that two conditions have been confused and that some of the red-cell forms are of the nature of these peculiar polar bodies. Kasarinoff also draws attention to the spherical plasmocytes of birds' blood. They are portions torn off the plasma of thrombocytes and may contain remnants of the polar bodies at their centres and thus simulate human blood-platelets.

Goodall's paper ('Journal of Pathology and Bacteriology,' 1910), giving an account of the red and white blood-cells of certain animals, will be found useful by laboratory workers in the Tropics ; though it must not be forgotten that the state of the blood in hot countries may differ very considerably from that in temperate climates. We have no tropical standards to guide us as regards the lower animals. Conditions met with in smears from such organs as the spleen and liver are not dealt with.

Mention is made of a few recent and little-known discoveries of true hæmatozoa.

Taking human blood first, there is the ectoglobular vermiform parasite found by the brothers Sergent in Algeria—now named by Brumpt *Sergentella hominis*. It is something like the leucocytozoon of a bird in shape, being pointed at the ends and containing a large oblong nucleus at its centre. These parasites exhibited periodicity, disappearing from the blood in the evening, and may have caused the nausea and sweating from which their human host suffered. In some illustrations it looks not unlike Nuttall's buffalo spirochaete and Balfour's hartebeeste forms.

Note is taken of somewhat crescentic and vacuolated bodies found by Castellani and Willey in human febrile cases and by Castellani and Sturgess in *Bos indicus*.

Hoefer has found an unknown protozoon in a case of anæmia at Leipzig. The patient, a woman, had never been away from the neighbourhood of that city.

It stains like a malarial parasite, shows chromatin, occurs in the red cells as rings or as curved pyriform bodies, also as a large blue-staining mass, dotted with fine chromatin granules and rather like a benign tertian parasite, but devoid of pigment. In the plasma double, possibly dividing, free forms occur which may show two nuclei each, and are either pear- or somewhat spindle-shaped. There is also another free form something like a stout vermicule.

The possible occurrence of hæmogregarines in human blood of the type found in certain mammals must be borne in mind. The nature of the X-bodies of Howell and Horrocks is not known. They seem to be associated with febrile conditions and may be of a parasitic nature. Balfour has seen them once in the blood of a sick dog. When stained with Leishman's stain, the bodies were characterised by a faint capsule with a circular centre staining deep blue. In size they varied from as large as to one-eighth the size of a red corpuscle. These forms were most common, but others were seen:—*(a)* A small blue circular centre surrounded by four or more faint capsules concentrically arranged ; *(b)* two circular bodies, each having a dark blue central point surrounded by a light blue ring enveloped in one capsule, which appeared indented as if two capsules were in process of formation ; *(c)* similar to *(b)*, but the part surrounding the deep blue centre stained a deeper blue, and two indented capsules were seen ; *(d)* a dark blue central part, shaped like a crescent, containing a small circular body with a deep blue central point within the arms of the crescent. None of the bodies on the slide showed any signs of chromatin. Later, Balfour found them in human blood in a case of urticaria without fever. Major Howard Ensor has also come across them in a case of fever and in a splenic puncture from a case of supposed kala azar.

As regards parasites in animal blood, certain unidentified forms in frogs' blood claim attention. One form found in the red cells was described by Dutton, Tod, and Tobey. They appear as more or less spherical chromatin-staining bodies. Carini suggests that it represents an endoglobular stage in the life-history of a trypanosome, for he has found and figured such a condition working with frogs' blood in Brazil. At an early stage the small inclusions he describes resemble the forms of Dutton, Tod, and Tobey. The so-called cytamœbæ, at least in certain of their forms, which these latter also found in frogs' blood in West Africa, are even more like Carini's early trypanosome stages, and also closely resemble some of the "after phase" bodies in the blood of spirochætal infected fowls. Other cytamœbæ in the frog, however, approximate to the "bacilloid" and bacillary bodies also found by Carini in *Leptodactylus ocellatus*. Bacilloid forms are little oval blue-staining masses with a single central chromatin dot, spherical or star-shaped, or two granules, one at either end, or a splash of chromatin along their sides. They may be single or multiple, the latter being apparently division forms. Kruse first found the bacillary forms in frogs caught in the neighbourhood of Paris. Their frequent occurrence in the red cells of European frogs has been noted by Labbé, Laveran, Ziemann, and others. They look like little branches of bacilli irregularly arranged at one end of the erythrocyte and having no connection with the nucleus. They may have a beaded look, may appear like a tangled skein in vacuoloid space, or look like rods sticking out of a central dark-staining mass.

Carini's forms are shorter or thinner than those called *Bacillus krusei* and may be specifically distinct. So far, however, no definite conclusion has been come to about any of these bodies, though they are apparently undoubtedly parasitic. The same is probably true of the small spherical pink-staining (with Giemsa) bodies found in the red cells of the leather-jacket fish of the Australian seas. Multiple infection of the red cells was, however, never present—a point rather against their being protozoa. Another point to be borne in mind is that Probat, of Muansa, and Francis, of Texas, have found what seem to be the free spores of *Sarcosporidia* in the blood of animals.

Dr. Balfour directs attention to the uses of the 'Folia Hæmatologica'—a mine of wealth for the hæmatologis The hope is expressed that someone with time, leisure, and the necessary knowledge will produce an English work which may aid those with little time to read lengthy papers, who have to work both at human and animal blood, and who are constantly meeting with appearances which they either cannot explain or class vaguely as degenerations. This is a hope most English workers will heartily endorse.

Acknowledgment must be made to Dr. Balfour for the somewhat full abstract of his paper here given. Failing the much-needed book in English, his paper goes far to remedy the want—at least for those not of the elect among hæmatologists. Its full value can only be appreciated when considered together with the excellent coloured plates, wherein are illustrated nearly all the points dealt with.

H. M. H.

THE CRAGGS PRIZE.

This Prize owes its origin to the generosity of Sir John Craggs, M.V.O., who, on the occasion of the Dinner presided over by Mr. Joseph Chamberlain in 1899 in connection with the foundation of the School, announced his intention to provide £300 per annum for a Travelling Scholarship for three years. At the expiration of that period he continued his benefaction by providing annually a prize of £50 for the Student of the London School of Tropical Medicine who in competition shows evidence of having made the most valuable contribution to the knowledge of Tropical Medicine during the year.

The prize has been always eagerly competed for, and the work of deciding as to the merit of the papers sent in has often been a difficult one.

It is not too much to say that the action of Sir John Craggs gave a stimulus to the School that has very largely conduced to the continuous success that has attended its labours.

A perusal of the following memoranda in relation to the work of the successful Candidates and the knowledge that has accrued in the course of their investigations will convince, not only members of the profession, but the ordinary reader, of the vast benefit that has been conferred upon mankind by the establishment of this prize.

The first investigator to be appointed in connection with Sir John Craggs's gift was Dr. G. C. Low, a Student of the School, who, at a meeting of Lecturers and Teachers in the School held in May 1900, was unanimously elected Craggs Research Scholar, and forthwith arrangements were made for the research work which it was proposed to carry out during the three years of his Scholarship.

In the first instance it was decided to demonstrate without any possibility of doubt that malaria is carried from one patient to another by means of the mosquito, and accordingly it was decided to make an experiment in a malarious district. Dr. Louis Sambon, one of the Teachers in the Tropical School, and Mr. A. Engel Terzi, an artist, were associated with Dr. Low for this purpose, and the summer of 1900 they spent in the Roman Campagna.

The experiment conclusively demonstrated the ability to live in a notoriously malarious area without acquiring infection, provided the body is protected from the bites of malaria-carrying mosquitoes: this was attained by sleeping in a properly constructed mosquito-proof house, the experimenters retiring to the house half an hour before sunset and remaining in it till one hour after sunrise.

At the same time mosquitoes were infected with malaria in Italy and sent home to England. Two volunteers in London, the late Dr. P. T. Manson and G. Warren, when bitten by these, acquired malaria, thus demonstrating the accuracy of the theory propounded by Sir Patrick Manson. At the end of the summer the expedition returned to England, and on the 26th December, 1900, Dr. Low left for the West Indies to investigate filarial and other tropical diseases. He visited St. Lucia, Dominica, Barbados, St. Vincent, St. Kitts, Grenada, Trinidad, and British Guiana. In addition to estimating the prevalence of filariasis in these different areas, experimental work on the life-history and other points in connection with these parasites were worked out. Later, at the request of the Governor of Barbados, Dr. Low instituted sanitary courses and lectures on the destruction of mosquitoes as a means of freeing the West Indian Islands from yellow fever, filariasis, and other diseases.

A series of lectures of a similar nature were also delivered in St. Kitts. In December 1901, an outbreak of yellow fever having appeared in St. Lucia, Dr. Low returned to that island and cooperated with the army doctors in stamping out the disease.

Further useful work on mosquito destruction by fumigating and destruction of breeding-places was conducted here.

Dr. Low then proceeded to British Guiana, and visited many of the tribes of aboriginal Indians who inhabit the primeval forest. Here a peculiar form of filariasis from which they suffer was investigated. After finishing this work Dr. Low returned to England in April 1902, and almost immediately started for Uganda as the leader of the first Sleeping Sickness Commission, a Commission organized by the Government and the Royal Society. At that time there was an idea that a filaria, viz. *Filaria perstans*, might be the cause of this disease, and therefore it was essential to determine whether this was so or not. Dr. Low's special part of the work was to determine this point, and he came to the conclusion, after a prolonged study of the question, that the filaria had nothing to do with the etiology of sleeping sickness.

Dr. Low returned to England in December 1902, and took up his duties as the Superintendent of the London School of Tropical Medicine on the 15th January, 1903. He relinquished the remainder of the Craggs Scholarship at that date, and Sir John Craggs then decided to abolish the Scholarship, substituting in its place the Craggs Prize, a prize awarded annually for the best original piece of work by past or present Students of the London School of Tropical Medicine.

Since that date many distinguished men have received this prize. In 1903 Dr. A. Castellani was successful with his work on the trypanosome as the cause of sleeping sickness. He was the first to find trypanosomes in that disease, a discovery which entirely altered our conception of that deadly malady.

Dr. Castellani is now stationed in Ceylon. There he has done a great deal of work on the causation of Yaws, a disease very prevalent in many tropical parts, and has described the causative agent of that disease. He has also done much to differentiate the various tropical skin-affections and to isolate the causative organisms of the same.

In 1904 Dr. J. Catto became Craggs Prizeman and discovered a new *Schistosomum*, a parasitic worm now known to be of great importance in China and Japan. In honour of Dr. Catto, Professor Blanchard, of Paris, called this the *Schistosomum cattoi*, but it was discovered that just before this date a Japanese scientist had seen and described the same parasite and named it *Schistosomum japonicum*, which name has the priority. All honour, however, is due to Dr. Catto, who quite independently made his discovery.

In 1905 the Committee decided to halve the prize, two papers of equal merit from Dr. Andrew Balfour, of Khartoum, and Dr. Philip Ross, of Uganda, having been received. Dr. Balfour submitted a paper describing his observations on a parasite known as a Hæmogregarine discovered by him in the blood of the jerboa or desert-rat of the Soudan. He also was able to demonstrate a considerable portion of its life-history. Dr. Balfour's parasite, named the *Hæmogregarina balfouri*, was one of the earliest of this type of parasite to be discovered in mammalian blood. Dr. Balfour is at present the

Director of the Wellcome Research Laboratory at Khartoum, and has done much to advance our knowledge of tropical pathology. He has also devoted much attention to hygiene, and has instituted many hygienic reforms.

Dr. Philip Ross, in conjunction with Dr. Milne of Uganda, made the very important discovery that the cause of African tick-fever was a spirochæte demonstrable in the blood of people affected with this complaint, a piece of original work of the first importance. It was felt that the best way to recognise Dr. Ross's work was to allot half the prize to him.

In 1906 Fleet-Surgeon Bassett Smith, C.B., R.N., was awarded the prize for his able researches in Undulant Fever, a fever which has caused endless trouble to those in authority in the Army and Navy, but which, by the work of a subsequent Commission, has finally been rendered almost harmless, the real transmitting agent—goats' milk—having at last been discovered.

In the years 1907 and 1910 no awards of the prize were made, as the papers submitted were not deemed to be of sufficient merit to warrant the prizes being given. Sir John Craggs, however, generously approved of the amount of his prize being allocated to the furtherance of research in various parts of the world. This has enabled grants to be made to individual workers, who have been termed Craggs Investigators.

Among these are :—

1. Dr. B. M. Wilson, of Fiji, for his investigations into the nature and propagation of Filariasis.
2. Dr. H. Bayon for researches on Leprosy.
3. Dr. R. Howard for investigation of the various insect carriers of disease in the University Mission Settlement in Nyasaland.
4. Dr. Cochran, to enable him to pursue his researches on Kala Azar in China, with special reference to the presence of *Leishmania* in superficial lymphatic glands. The results already are enough to show that the condition is sufficiently common to be of great diagnostic value and not a rarity, as was believed when his discovery was first announced.

In the year 1908 the prize was divided between Dr. A. E. Horn, of the West African Medical Service, and Dr. W. Fletcher, of the Federated Malay States Medical Service.

Dr. Horn contributed an account of a very able investigation, carried out in the face of great difficulties, into an epidemic of cerebro-spinal meningitis in West Africa. Dr. Fletcher's observations, carried out in a true scientific spirit, did much to show that the consumption of certain kinds of rice was responsible for beri-beri, a disease claiming numerous victims and affecting to an enormous extent the output of labour. He thus showed that the hypothesis of the cause of the disease as put forward by Braddon was probably correct.

In 1910 Dr. G. Turner, of Johannesburg, was the recipient of the prize. He presented an excellent account of his observations on disease caused by the Bilharzia worm in South Africa. He was able to show that the eggs of this worm can be found on occasions in the lungs, an observation not previously recorded, and also described the grave organic changes the eggs give rise to in these organs.

In 1911 the prize was again divided, this time between Dr. H. Fraser, the

Director of the Kuala Lumpur Laboratory, and Dr. A. T. Stanton, Bacteriologist in the same Laboratory. These old Students in collaboration have produced a work of the greatest importance on 'The Etiology of Beri-Beri,' a disease already dealt with by a prize-winner—Dr. W. Fletcher—in 1908.

There is still in hand, on account of the two years when the prize was not awarded, a sum of £40, and this remains at disposal for the appointment of investigators in any subject that the School may consider of sufficient importance.

STANLEY EXPEDITION: FIRST REPORT.

As our readers are aware, it is proposed that Reports embodying the results of the work of our expeditions in the Tropics should be published from time to time, and where too long for insertion in the Journal, should be considered as supplements and published in such a size that they can be conveniently bound up with the Journal if so desired. Dr. P. H. Bahr was in charge of the expedition to Fiji, and the main results of this expedition, the funds for which were provided by the Stanley family in memory of the late Hon. Edward John Stanley, are embodied in two excellent reports shortly to be published by Messrs. Witherby & Co., 326 High Holborn, W.C. The first of these, 'Filariasis and Elephantiasis in Fiji,' will be published about the middle of February, price 6*s*. It is not only an account of the conditions in Fiji, but contains many new and original observations of great importance. It is profusely illustrated. To all interested in the subject the report will be welcome, as much new light is thrown on many of the problems connected with filariasis.

The second report, 'Dysentery in Fiji,' of which Messrs. Witherby & Co. are also the publishers, it is hoped will be ready in March.

Application for these reports should be made to the publishers.

I.—ENTOMOLOGICAL NOTES.

By A. ALCOCK, C.I.E., F.R.S.

Epitome of the Species of Blood-sucking *Muscidæ*, *Glossina* excepted.

By SOPHIA L. M. SUMMERS, M.A., B.Sc.,
Carnegie Scholar of Aberdeen University.

The blood-sucking *Muscidæ* have of late years received considerable attention, for although they are not known—the species of *Glossina* excepted—to have any special predilection for man, or to be necessary agents in spreading the specific infection of any human disease, yet they are rightly regarded as being potentially dangerous in this respect. Austen has treated the African species with his usual ability; and Bezzi, in the 'Archives de Parasitologie' for 1911, has given a learned account of the entire group from the entomological standpoint; but there still appears to be room for a summary of all the species known, arranged to suit the convenience of the student of human and veterinary pathology.

Among *Muscidæ* the blood-sucking species can usually be recognised by the rigid, chitinous, non-retractile, and more or less tapering proboscis. Occasionally, however, as in *Philæmatomyia*, the proboscis is fully retractile and to a considerable extent fleshy, and ends in large fleshy labella, but even in this case it is furnished with strong teeth that can cut through skin. In other respects, except sometimes in certain details of the wing-venation and of the feathering of the arista, most of them have a general resemblance to the common house-fly.

The genus *Glossina*, which is here excluded from consideration, differs from all other blood-sucking *Muscidæ* in characters which some entomologists consider to be of "family" value. In *Glossina* the shaft of the proboscis is particularly slender; the maxillary palps are long and slender, and they form a sheath for the proboscis, a condition which, among other blood-sucking *Muscidæ*, is approached only by *Lyperosia*; the individual hairs of the arista are pinnate; the fourth longitudinal vein of the wings has a double curve, the terminal curve, or bend, taking-off at the junction with the posterior cross-vein; the abdomen is composed of seven distinct segments; and in the male the hypopygium is a large and complicated organ of peculiar shape. *Glossina* also differs from other blood-sucking *Muscidæ* in the pose of its wings when at rest, and stands apart from all other *Muscidæ* in its "pupiparous" method of reproduction.

In all the other blood-sucking *Muscidæ*—those that form the subject of this paper—the shaft of the proboscis is not greatly attenuated; the maxillary palps, if slender, are short, and if long are more or less spatulate; the hairs of the arista are simple; the fourth longitudinal vein runs a normal course, and curves or bends forwards at a point much beyond the junction with the posterior cross-vein; the abdomen appears to consist of only four segments; and there is nothing peculiar either in the pose, or in the method of reproduction.

TABLE OF GENERA.

	Proboscis entirely chitinous with attenuated labella	1.
	Proboscis partly chitinous, partly fleshy, with large fleshy labella	*Philæmatomyia.*
1.	Arista feathered dorsally only	2.
	Arista feathered dorsally and ventrally	3.
2.	Maxillary palps slender, less than half the length of the proboscis. Third longitudinal vein with some bristles on its proximal part	*Stomoxys.*
	Maxillary palps more or less spatulate, almost as long as the proboscis, or at any rate very much more than half its length	4.
4.	Proboscis long and tapering; the first posterior cell widely open; third longitudinal vein without bristles	*Lyperosia.*
	Proboscis short and stumpy; the first posterior cell narrowly open; third longitudinal vein with some bristles proximally	*Stygeromyia.*
3.	First posterior cell narrowly open; third longitudinal vein without bristles	*Hæmatobosca.*
	First posterior cell widely open	5.
5.	Third longitudinal vein with some bristles proximally	*Hæmatobia.*
	Third longitudinal vein without bristles	*Bdellolarynx.*

STOMOXYS, Geoffroy.

Stomoxys, Geoffroy, Hist. abrégie des Insectes qui se trouvent aux environs de Paris, ii. p. 538, 1762; Grünberg, Die blutsaugenden Dipteren, p. 155, 1907; Bezzi, Zeits. f. Hymenopt. und Dipt. vii. 1907, p. 413, and Archiv. de Parasitol. xv. 1911, p. 124; Austen, African Blood-sucking Flies, 1909, p. 141; Brunetti, Rec. Ind. Mus. iv. 1910, p. 66; Alcock, Entomology for Medical Officers, p. 158.

See also Hansen, in Austen's Monograph of the Tsetse-flies, p. 105, pl. viii., ix. (mouth-parts); Giles in Journ. Trop. Med. April to July 1906, and Tulloch in Roy. Soc. Proc. B, lxxvii. 1906, p. 523 (internal anatomy); Newstead in Journ. Econ. Biol. 1906 (life-history); Newstead & Stephens, Ann. Trop. Med. i. 1907, p. 171 (anatomy of proboscis).

Front narrower in the male than in the female; arista of three segments, feathered on the dorsal (anterior) edge only; proboscis long, tapering, chitinized in all its extent, non-retractile; palpi slender, very short, less than half the length of the proboscis; 4th longitudinal vein curved (not abruptly bent) so as merely to narrow the 1st posterior cell distally; 3rd longitudinal vein bristly at its proximal end.

Geographical Distribution. 23 species, not including several doubtful proposals, are at present known, one of which (*S. calcitrans*) is found in all parts of the world. The remaining 22 are distributed as follows: Africa 13 species, Asia 12 species, 3 of which are common to Africa.

The species of *Stomoxys* very much resemble house-flies in general appearance, but can easily be distinguished by the hard, shiny, tapering, non-retractile proboscis, and by the form of the 4th longitudinal vein of the wing, which is not *abruptly* bent forwards. Though they are exceedingly troublesome to animals they sometimes attack man and may be found in houses.

A.—EUROPEAN SPECIES.

1. Stomoxys calcitrans, L.

Conops calcitrans, Linnæus, Syst. Nat. ed. x. 1758, i. p. 604.
Stomoxys calcitrans, Schiner, Faun. Austr., Fliegen, i. p. 578, 1862; Austen, British Blood-sucking Flies, pl. 29, and African Blood-sucking Flies, p. 142, pl. xiii. fig. 102.
For list of synonyms of this species see Catalogue of Palæarctic Diptera, iii. p. 611, also Brunetti, Rec. Ind. Mus. iv. 1910, p. 69, and Bezzi, Archiv. de Parasitol. xv. 1911, p. 138.

This species can be recognised by the round spots on the dorsum of the abdomen—three on the 2nd and three on the 3rd segment. The face is yellowish white, and the front is wide both in male and female. Of the four longitudinal black stripes on the scutum (which are present in all the species) the two median ones are slightly convergent anteriorly. The legs are black, except for the knees, which are reddish brown.

Austen in his 'African Blood-sucking Flies' has collected numerous references on the subject of this common species as a carrier of disease among domestic animals.

B.—ASIATIC SPECIES.

Fourteen species have been reported from Asia, namely *bengalensis*, Picard, *brunnipes*, Grünb., *calcitrans*, L., *dacnusa*, Speiser, *indica*, Picard, *limbata*, Austen, *nigra*, Macquart, *oblongopunctata*, Brunetti, *plurinotata*, Bigot, *prattii*, Summers, *pulla*, Austen, *pusilla*, Austen, *sitiens*, Rondani, *triangularis*, Brunetti; but one of them, namely *S. bengalensis*, Picard, is probably only a synonym of *S. nigra*, Macquart, and another, namely *S. plurinotata*, Bigot, is possibly identical with *S. calcitrans*, L., so that the valid species at present known from Asia are more probably twelve in number, three of the twelve being common to Africa.

(*a*) Legs mainly black, abdomen spotted.

1. Stomoxys calcitrans, L.

Vide supra.

2. Stomoxys sitiens, Rondani.

Rondani, Ann. Mus. Genov. iv. 1873, p. 288; Brunetti, Rec. Ind. Mus. iv. 1910, pp. 68 & 75; Summers, Ann. Mag. Nat. Hist. (8) viii. 1911, p. 237.

Like *S. calcitrans*, but smaller, the face shiny white, and the front in the male much narrower, being one-eighth or less the width of the head.

This species, described by Rondani from Abyssinia, is stated by Brunetti (Rec. Ind. Mus. iv. 1910, p. 75) to occur in Calcutta, Penang, and Rangoon.

Stomoxys plurinotata, Bigot.

Bigot, Bull. Soc. Zool. France, xii. 1887, p. 593; Brunetti, Rec. Ind. Mus. iv. 1910, pp. 67 & 73; Summers, Ann. Mag. Nat. Hist. (8) viii. 1911, p. 237.

This is probably the same as *S. calcitrans*, L., but is said to differ in having "4 large oblong-elongate spots or interrupted stripes," instead of the usual uninterrupted stripes, on the scutum.

Loc. Ceylon.

3. STOMOXYS OBLONGOPUNCTATA, Brunetti.

Brunetti, Rec. Ind. Mus. iv. 1910, p. 73; Summers, Ann. Mag. Nat. Hist. (8) viii. 1911, p. 237.

Resembles *S. calcitrans* in having black legs and a spotted abdomen, but the abdominal spots are transversely oblong, not round, and the front of the male is narrower—only one-fifth the breadth of the head.

Loc. Assam and Kuala Lumpur.

4. STOMOXYS TRIANGULARIS, Brunetti.

Brunetti, Rec. Ind. Mus. iv. 1910, p. 77; Summers, Ann. Mag. Nat. Hist. (8) viii. 1911, p. 237.

Also resembles *S. calcitrans*, but the spots of the abdomen are large and triangular, sometimes almost meeting in the middle line, and the front of male is narrower—only one-fifth of the breadth of the head. It is a smaller species than *S. oblongopunctata.*

Loc. S. India and Kuala Lumpur.

(*b*) Legs mainly black. Abdomen cross-banded.

5. STOMOXYS NIGRA, Macquart.

Macquart, Dipt. Exot. Suppl. iv. 1850, p. 239; Austen, African Blood-sucking Flies, 1909, p. 153; Summers, Ann. Mag. Nat. Hist. (8) viii. 1911, p. 237.

Agrees with *S. calcitrans* in having the legs mainly black, but differs in having cross-bands on the abdomen, due to the black colouring of the after edges of the first three segments.

An African species which is found in the Malay States.

STOMOXYS BENGALENSIS, Picard.

Picard, Bull. Soc. Ent. Fr. 1908, p. 21; Brunetti, Rec. Ind. Mus. iv. 1910, p. 79; Summers, Ann. Mag. Nat. Hist. (8) viii. 1911, p. 238.

I have not seen this species, but from the description I consider that it is identical with *S. nigra*, Macquart.

Loc. Bengal.

(*c*) Legs mainly brown. Abdomen not spotted but often cross-banded.

6. STOMOXYS BRUNNIPES, Grünberg.

Grünberg, Zool. Anz. xxx. 1906, p. 89; Brunetti, Rec. Ind. Mus. iv. 1910, p. 74; Summers, Ann. Mag. Nat. Hist. (8) viii. 1911, p. 238.

Differs from *S. calcitrans* in having (*a*) the abdomen brownish yellow, with broad black cross-bands as in *S. nigra*, (*b*) brown tibiæ and tarsi, and (*c*) the front of the male extremely narrow—one-eighth the breadth of the head.

It is an African species which, according to Bezzi (Archiv. de Parasitol. xv. 1911, p. 138), also occurs in Java.

7. STOMOXYS INDICA, Picard.

Picard, Bull. Soc. Ent. Fr. 1908, p. 20; Brunetti, Rec. Ind. Mus. iv. 1910, p. 77; Summers, Ann. Mag. Nat. Hist. (8) viii. 1911, p. 237.

A *small* species, otherwise resembling *S. brunnipes*, from which, however, it can be distinguished by having the femora, as well as the tibiæ and tarsi, brown, and by the front in the male being a perfectly straight line.

Loc. Bengal.

8. STOMOXYS PULLA, Austen.

Austen, Ann. Mag. Nat. Hist. (8) iii. 1909, p. 294; Brunetti, Rec. Ind. Mus. iv. 1910, p. 80; Summers, Ann. Mag. Nat. Hist. (8) viii. 1911, p. 237.

A small species with the legs all brown. It differs from *S. indica* in being dark, with the abdomen blotchy, not cross-banded. Its characteristic feature in the male is the fore tarsus, which has the first joint fringed on the inside with a row of hairs of equal length.

Loc. India.

9. STOMOXYS PUSILLA, Austen.

Austen, Ann. Mag. Nat. Hist. (8) iii. 1909, p. 293; Brunetti, Rec. Ind. Mus. iv. 1910, p. 80; Summers, Ann. Mag. Nat. Hist. (8) viii. 1911, p. 237.

Also a small species with brown legs. The abdomen is cross-banded on the third segment, the second segment being entirely clove-brown. The front of the male is comparatively wide—one-fourth the breadth of the head.

Loc. India.

10. STOMOXYS LIMBATA, Austen.

Austen, Ann. Mag. Nat. Hist. (8) iii. 1909, p. 292; Brunetti, Rec. Ind. Mus. iv. 1910, p. 78; Summers, Ann. Mag. Nat. Hist. (8) viii. 1911, p. 237.

Also a small species with brown legs and cross-banded abdomen, but the front in the male is constricted in the middle, not straight.

Loc. "India" and Kuala Lumpur.

11. STOMOXYS PRATTII, Summers.

Summers, Ann. Mag. Nat. Hist. (8) viii. 1911, p. 238.

A large species with brown legs—the tibiæ and tarsi being pale yellow—and abdomen cross-banded. Front of the male one-sixth the breadth of the head.

Loc. Kuala Lumpur.

12. STOMOXYS DACNUSA, Speiser.

Speiser, Zool. Anz. xxxiii. 1908, p. 666; Brunetti, Rec. Ind. Mus. iv. 1910, p. 81; Summers, Ann. Mag. Nat. Hist. (8) viii. 1911, p. 237.

It is difficult to estimate the value of this "species," as it was described from a single damaged female specimen.

"Front one-third the breadth of the head. Legs pale brown. Abdomen grey. Thorax and first segments of abdomen cannot be described as they are covered with blood."

Loc. Annam.

C.—AFRICAN SPECIES.

Eighteen species have been described from Africa, but two of them (namely *S. glauca* and *S. lafonti*) are regarded as synonymous with *S. nigra*, one (*S. sellata*) with *S. brunnipes*, and one (*S. korogwensis*) with *S. calcitrans*, so that the number of species that are probably valid is fourteen.

(*a*) Legs mainly black, abdomen spotted.

1. Stomoxys calcitrans, L.

Vide supra (European species).

Stomoxys korogwensis, Grünberg.

Grünberg, Zool. Anz. xxx. 1906, p. 88.

This species is considered by Bezzi (Archiv. de Parasitol. xv. 1911, p. 138) to be a synonym of *S. calcitrans*, L.

2. Stomoxys sitiens, Rondani.

Vide supra (under Asiatic species).

3. Stomoxys griseiceps, Becker.

Becker, Berlin Mitt. Zool. Mus. iv. 1908, p. 195.

Like *S. calcitrans*, L., but the abdomen is black with a brown median line, and the spots are greyish brown.
Loc. Madeira.

(*b*) Legs mainly black, abdomen not spotted.

4. Stomoxys nigra, Macquart.

Vide supra (under Asiatic species).

Stomoxys glauca, Grünberg.

Grünberg, Zool. Anz. xxx. 1906, p. 88.

This species is stated by Austen in 'African Blood-sucking Flies,' 1909, p. 153, to be synonymous with *S. nigra*, Macq.

Stomoxys lafonti, Picard.

Picard, Bull. Soc. Ent. Fr. 1907, p. 28.

This is described as a new species from the Mauritius, but is considered by Bezzi (Archiv. de Parasitol. xv. 1911, p. 138) to be a synonym of *S. nigra*, Macq.

5. Stomoxys bouvieri and var. clara, Roubaud.

Roubaud, Ann. Inst. Pasteur, xxi. 1907, p. 667.

Like *S. nigra*, Macq., but is larger and the stripe on the thorax is broader. There is a variety of this species, namely *clara*, which has the wings and halteres colourless both in male and female, whereas in *S. bouvieri* the wings and halteres in the male are smoky.
Loc. Congo.

6. Stomoxys inornata, Grünberg.

Grünberg. Zool. Anz. xxx. 1906, p. 90; Picard, Bull. Soc. Ent. Fr. 1907, p. 312.

A dark species, the abdomen being uniformly black. The front in the male is very narrow—about one-twelfth the breadth of the head. Legs black with brown knees.
Loc. Africa.

7. Stomoxys omega, Newstead.

Newstead, Ann. Trop. Med. i. 1907, p. 87.

Characterised by the colouring of the scutum, which is bluish grey with a black ∿-shaped device—very distinct in the male but not so distinct in the female. Abdomen black. Legs black with brown knees. Front very narrow.
Loc. Congo.

(c) Legs not mainly black.

8. Stomoxys varipes, Bezzi.

Bezzi, Rend. Ist. Lomb. (2) xl. 1907, p. 446.

Seems to differ from *S. calcitrans*, L., mainly in the colour of the femora, which are more or less infuscated, and of the tibiæ, which are yellow. The breadth of the front is not stated. The spots of the abdomen are elongate.
Loc. Erythræa.

9. Stomoxys intermedia, Roubaud.

Roubaud, Ann. Inst. Pasteur, xxi. 1907, p. 666.

This species was described from the female only. Thorax grey with a distinct median line. Abdomen yellowish grey with brownish-black bands on the first three segments. Tibiæ and tarsi brownish.
Loc. Congo.

10. Stomoxys bouffardi, Picard.

Picard, Bull. Soc. Ent. Fr. 1907, p. 27.

Front very broad, half the breadth of the head. Legs brown, the trochanter, knees, and tibiæ red, the latter sometimes black at the tip; second segment of the abdomen with two grey hairy bands in the middle.
Loc. Soudan.

11. Stomoxys bilineata, Grünberg.

Grunberg, Zool. Anz. xxx. 1906, p. 89.

Differs from *S. nigra*, Macq., in having the tibiæ of the first two pairs of legs and the knees of the third pair light brown; the second and third segments of the abdomen having brown bands instead of black, and the dark stripes of the scutum disappearing behind the suture.
Loc. East Africa.

12. Stomoxys brunnipes, Grünberg.

Vide supra (under Asiatic species).

STOMOXYS SELLATA, Grünb.

Grünberg, Zool. Anz. xxx. 1906, p. 90.

This species is considered by Bezzi (Archiv. de Parasitol. xv. 1911, p. 138) to be synonymous with *S. brunnipes*, Grünb.

13. STOMOXYS TÆNIATUS, Bigot.

Bigot, Bull. Soc. Zool. Fr. xii. 1887, p. 594.

Like *S. brunnipes*, Grün., but is larger; its legs are all a uniform pale yellow; and the front is much wider, being one-fourth to one-fifth the breadth of the head, while in *S. brunnipes* it is only one-eighth the breadth of the head.
Loc. Natal.

14. STOMOXYS OCHROSOMA, Speiser.

Speiser, Kilimandjaro-Meru Exp. Bd. 2; 10, v. 1910, p. 162.

The colour of this species is distinctive. It is golden yellow. The head is black. Proboscis is shiny yellow with black tip. Legs golden yellow with dark brown tarsi. Abdomen yellow.
Loc. Kilimandjaro.

D.—AMERICAN SPECIES.

STOMOXYS CALCITRANS (L.).

Vide supra.

Is a well-known species in both North and South America, and also in the West Indies.

E.—AUSTRALIAN SPECIES.

STOMOXYS CALCITRANS (L.).

Vide supra.

Has been reported in many parts of the Australian Region, from Australia to Fiji and Hawaii.

HÆMATOBIA, Rob.-Des.

Hæmatobia, Robineau-Desvoidy, Essai sur les Myodaires, 1830, p. 388; Bezzi, Zeits. f. Hymenopt. u. Dipt. vii. 1907, p. 416, and Archiv. de Parasitol. xv. 1911, p. 126; Brunetti, Rec. Ind. Mus. iv. 1910, p. 63; Alcock, Medical Entomology, 1911, p. 159.

Bezzi (*loc. cit.*) has discussed the synonymy of the genus and has given reasons for maintaining the generic name *Hæmatobia*.

Differs from *Stomoxys* (1) in the form and proportions of the maxillary palpi, which are spatulate and nearly as long as the proboscis; (2) in having hairs on the ventral (posterior) as well as on the dorsal (anterior) edge of the arista; and (3) in having the first as well as the third longitudinal vein bristly at the proximal end. The body also is more robust, the head is broader and more squat, and the proboscis is somewhat shorter and stouter than in *Stomoxys*.

Geographical Distribution. Europe, India, and North America.

The species of *Hæmatobia*, like those of *Stomoxys*, much resemble house-flies in general appearance. They attack animals and are not found in houses.

A.—EUROPEAN SPECIES.

One species is known from Europe, namely *Hæmatobia stimulans*, Meig.

1. HÆMATOBIA STIMULANS.

Siphona stimulans, Meigen, Syst. Beschr. Europ. Zweifl. Ins. iv. 1824, p. 161.
Hæmatobia stimulans, Austen, British Blood-sucking Flies, 1906, p. 59; Brunetti, Rec. Ind. Mus. iv. 1910, p. 64; Bezzi, Archiv. de Parasitol. xv. 1911, p. 126.

"Face white: in the male the eyes are almost in contact, in the female the front is broad. Antennæ brown. Proboscis somewhat longer than the head, palpi reddish yellow, club-shaped. Thorax and abdomen ash-grey. Thorax with black longitudinal stripes. Abdomen with black spots and a straight longitudinal stripe. Wings in male brownish, in the female colourless. Legs black."

B.—ASIATIC SPECIES.

There are only two species described from Asia—*Hæmatobia sanguisugens*, Austen, and *Hæmatobia rufipes*, Brunetti.

2. HÆMATOBIA SANGUISUGENS, Austen.

Austen, Ann. Mag. Nat. Hist. (8) iii. 1909, p. 288; Brunetti, Rec. Ind. Mus. iv. 1910, p. 64.

"Length five and a half to six mm. Colour brownish grey with clove-brown markings. Dorsum of thorax with two pairs of clove-brown longitudinal stripes (outer stripe broader and widely interrupted at transverse suture), and a less sharply defined median stripe: dorsum of abdomen with a clove-brown median longitudinal stripe, extending from front margin of second to beyond middle of fourth segment, narrowly interrupted before the hind margins of the second and third segments, a pair of transversely elongate dusky blotches on the first segment, a pair of large clove-brown spots on the second segment, and a pair of similar but smaller spots on the third segment. Wings slightly infuscated, light sepia-coloured. Legs clove-brown, bases of tibiæ ochraceous buff."

Loc. India.

3. HÆMATOBIA RUFIPES, Brunetti.

Brunetti, Rec. Ind. Mus. iv. 1910, p. 65.

This "species," described from only two females, is considered by Bezzi, with whom I agree, to be probably the female of *H. sanguisugens*, Austen.

"Length four to five mm. Colour yellowish grey. Scutum with a broad median grey stripe from anterior margin to beyond the middle: this stripe edged with a dark brown but not sharply defined line. Abdomen with a narrow median black stripe, each segment mainly occupied by a pair of large blackish spots of irregular shape. Legs reddish yellow. Fore femora, tips of hind femora, and the tarsi blackish."

Loc. Himalayas and Nepal.

C.—AMERICAN SPECIES.

One species has been described from N. America, namely *Hæmatobia alcis*, Snow, and it is considered by Bezzi to be a *Lyperosia*. As, however, it is stated to have one or two hairs on the posterior (ventral) edge of the arista, it may, for the present, be left as a *Hæmatobia*.

4. HÆMATOBIA ALCIS, Snow.

Snow, Canad. Ent. xxiii. 1891, p. 89.

"Length four to six mm. Thorax grey with a yellowish tinge. Scutum with two very distinct black median stripes and an interrupted stripe on either side. Abdomen yellowish grey with a brownish black median stripe, and a pair of dusky spots on the second and third and sometimes on the fourth segments. Legs brownish black. Wings hyaline, base orange-yellow."

HÆMATOBOSCA, Bezzi.

Bezzi, Zeitschr. f. Hymenopt. u. Dipt. vii. 1907, p. 414, and Archiv. de Parasitol. xv. 1911, p. 127; Brunetti, Rec. Ind. Mus. iv. 1910, p. 61; Alcock, Entomology for Medical Officers, 1911, p. 159.

Like *Hæmatobia*, Rob.-Desv., but has no bristles on the first and third longitudinal veins, *and has the fourth longitudinal vein almost as strongly bent as it is in Musca*, so as to leave the first posterior cell only very narrowly open.

Geographical Distribution. Southern Europe and China.

1. HÆMATOBOSCA ATRIPALPIS, Bezzi.

Hæmatobia atripalpis, Bezzi, Bull. Soc. Ent. Ital. xxvii. 1895, p. 60, and Zeitschr. f. Hymenopt. u. Dipt. vii. 1907, p. 414.

Thorax indistinctly striped ("vix nigrolineatus"); palps very black; abdomen grey except for a longitudinal black stripe; knees yellow, hind tibiæ ciliated both internally and externally in all their length; wings yellowish at base, 1st posterior cell much narrowed at apex.

Loc. Southern Europe.

2. HÆMATOBOSCA PERTURBANS, Bezzi.

Siphona perturbans, Bezzi, Rendic. Istit. Lomb., Milano, (2) xl. 1907, pp. 433-460.

"Grey: thorax with four longitudinal yellow stripes, less distinct in ♀. Abdomen with no spots or with indistinct yellow transverse spots; palps reddish brown; legs in both sexes black, knees yellow, arista with one hair on the ventral side; base of the wings yellow, no bristles, 1st posterior cell narrow at the apex. Length of body 3½–4½ mm. Length of wings 3½–4½ mm."

Loc. Tang San, China.

BDELLOLARYNX, Austen.

Austen, Ann. Mag. Nat. Hist. (8) iii. 1909, p. 290; Brunetti, Rec. Ind. Mus. iv. 1910, p. 62; Bezzi, Archiv. de Parasitol. xv. 1911, p. 123; Alcock, Entomology for Medical Officers, 1911, p. 159.

Like *Hæmatobia*, R.-D., but has no bristles on the first and third longitudinal veins of the wing.

"Small, stoutly-built, thick-set flies. Closely allied to *Hæmatobia*, Rob.-Desv., but distinguishable as follows :—No sexual colour-dimorphism (unless it be in the wings) ; front, facial angles, and anterior margin of buccal cavity less prominent ; jowls descending considerably behind, instead of nearly horizontal, but the basi-occipital region much less protuberant ; bristles below facial angles small and fine, instead of relatively coarse ; in ♂, front wider, with upper inner margins of the eyes more nearly parallel, less approximate in the middle of front ; arista feathered above with considerably longer hairs, and below with about six fairly long hairs ; first and third longitudinal veins entirely bare, without bristles at the base."

Only one species is known, and it is reported from India and Ceylon.

1. Bdellolarynx sanguinolentus, Austen.

Austen, Ann. Mag. Nat. Hist. (8) iii. 1909, p. 290; Brunetti, Rec. Ind. Mus. iv. 1910, p. 62.

"Length $4\frac{3}{4}$–5 mm. ♂; ♀ $3\frac{1}{2}$–$4\frac{1}{4}$ mm. Mouse-grey, clothed with short black hair ; palpi buff ; dorsum of thorax with a pair of extremely narrow, widely separated, parallel, longitudinal, admedian, blackish stripes, a somewhat triangular blackish mark extending backwards from inner end of each humeral callus, and an elongate blackish streak in a line with latter mark behind transverse suture ; dorsum of abdomen with a narrow, interrupted, longitudinal, median, clove-brown stripe, and, on second and third segments, paired, transverse, roughly triangular, clove-brown blotches, the pair on the second segment especially large ; wings hyaline or tinged with tawny olive in ♂, hyaline in ♀ : femora greyish clove-brown, extreme tips ochraceous buff, one or more pairs sometimes more or less brownish except at base, tarsi dark brown."

Lyperosia, Rondani.

Rondani, Dipt. Ital. Prodromus, i. 1856, p. 93, also v. 1862, p. 230 ; Bezzi, Rendic. Istit. Lomb., Milano, (2) xl. 1907, p. 456, and Zeitschr. f. Hymenopt. und Dipt. vii. 1907, p. 416, and Archiv. de Parasitol. xv. 1911, p. 128 ; Grünberg, Die blutsaugenden Dipteren, 1907, p. 160 ; Austen, African Blood-sucking Flies, 1909, p. 161 ; Brunetti, Rec. Ind. Mus. iv. 1910, p. 83 ; Alcock, Entomology for Medical Officers, 1911, p. 158.

Glossinella, Grünberg, Zool. Anz. xxx. 1906, p. 85, and Die blutsaugenden Dipteren, p. 161 ; *vide* Bezzi, Archiv. de Parasitol. xv. 1911, p. 136.

Differs from *Stomoxys* (1) in the form and proportions of the maxillary palps, which are as long as the long and slender proboscis, and are of fair breadth in all their extent, so as almost to form a sheath for the latter ; and (2) in the absence of bristles from the third longitudinal vein. The body also is slenderer than that of *Stomoxys*, and the species are, as a rule, very much smaller.

Geographical Distribution. Europe, Asia, Africa, N. America, and Australia.

The species of *Lyperosia* are the smallest of the blood-sucking *Muscidæ*. In colour they are dirty brown or dirty yellowish grey, with inconspicuous longitudinal markings on the scutum and abdomen. They usually attack animals, but sometimes trouble man, though they do not come into houses.

A.—EUROPEAN SPECIES.

Two species are known from Europe: (1) *Lyperosia irritans*, L., (2) *Lyperosia titillans*, Bezzi.

1. Lyperosia irritans, L.

Linnæus, Systema Naturæ, x. 1758, p. 1; Bezzi, Archiv. de Parasitol. xv. 1911, p. 133; Brunetti, Rec. Ind. Mus. iv. 1910, p. 89.

The following diagnosis of the species is verified from Bezzi's table in Archiv. de Parasitol. xv. 1911, p. 131:—

The sixth longitudinal vein is of good length; the first longitudinal vein reaches the costa beyond the level of the anterior cross-vein; eight to ten rather close-set hairs on the arista; in the last pair of legs in the male the tarsal segments are dilated distally (serrate), and the pre-apical bristle of the tibia is remarkably long.

2. Lyperosia titillans, Bezzi.

Bezzi, Arch. de Parasitol. xv. 1911, p. 136.

Distinguished from *L. irritans*, L., according to Bezzi, by the following characters: the sixth longitudinal vein extends only a short way beyond the anal cell; the first longitudinal vein reaches the costa inside the level of the anterior cross-vein; five or six rather distant hairs on the arista; in the last pair of legs the tarsi are not dilated and the preapical bristle of the tibia is of no remarkable length.

B.—ASIATIC SPECIES.

The number of valid species is probably three—all of them from the Oriental Region.

1. Lyperosia minuta, Bezzi.

Bezzi, Ann. Mus. Genov. (2) xii. 1892, p. 192, and Archiv. de Parasitol. xv. 1911, p. 131; Brunetti, Rec. Ind. Mus. iv. 1910, p. 84.

This species resembles *L. titillans*, Bezzi (*vide supra*), differing most conspicuously in the paler colouring of the legs and the distal narrowing of the first posterior cell. In the male also the front is decidedly broader.

An African species which also occurs in the Oriental Region.

2. Lyperosia schillingsi, Grünberg.

Grünberg, Zool. Anz. xxx. 1906, p. 86.

This species was described under the genus *Glossinella*, but Austen states (Bezzi, Zeitschr. Hymenopt. u. Dipt. vii. 1907, p. 415) that it is a *Lyperosia*: Bezzi, Archiv. de Parasitol. xv. 1911, p. 137.

Black species dusted with grey, bristles black; front bright grey and broad—about one-third the breadth of the head; first and second segments of the palps golden brown, the third black; arista with four or five hairs. Legs brown.

An African species also occurring in the Oriental Region.

3. Lyperosia exigua (Meijere).

(*Hæmatobia exigua*) Meijere, in P. Schat, "Verdere Medeleelingen over Surra," Mededeel. Proefstation, Oost-Java, 3e (3) xliv. (1903); Brunetti, Rec. Ind. Mus. iv. 1910, p. 86.

As in *L. irritans*, L., in the hind legs the tarsi are dilated, the tibia carries a long preapical bristle, and there are from eight to ten hairs on the arista. Legs mainly yellow. Front in male narrow—one-fifth to one-sixth breadth of head.

Loc. Java, India, Ceylon, China.

Lyperosia flavohirta, Brunetti.

Brunetti, Rec. Ind. Mus. iv. 1910, p. 89.

Differs from *L. exigua* (Meij.), according to its author's description, in its lighter colouring.

Is probably identical with *L. exigua* (Meij.).

"Lyperosia rufipalpis," Becker.

Becker, Denkschr. der math.-naturwiss. Klasse der k. Akad. der Wiss. Wien, lxxi. 1910, p. 148.

According to Bezzi (Archiv. de Parasitol. xv. 1911, p. 139) this is probably identical with *Stygeromyia maculosa*, Austen.

C.—AFRICAN SPECIES.

Eight species have been reported from Africa, two of which (*L. longipalpis*, Roubaud, and *L. pallidipes*, Roubaud) are considered to be synonyms.

1. Lyperosia minuta, Bezzi.

Vide supra (under Asiatic species).

Lyperosia longipalpis, Roubaud.

Roubaud, C. R. Soc. Biol. 1906, p. 896.

This species is considered by Austen ('African Blood-sucking Flies,' p. 160) to be synonymous with *L. minuta*, Bezzi.

Lyperosia pallidipes, Roubaud.

Roubaud, Ann. Past. Inst. xxi. 1907, p. 669.

Legs pale yellow, arista with six or seven hairs.

Bezzi considers that this species is the same as *L. minuta*, Bezzi.

2. Lyperosia schillingsi, Grünberg.

Vide supra (under Asiatic species).

3. Lyperosia potans, Bezzi.

Bezzi, Rendic. Istit. Lomb., Milano, (2) xl. 1907, p. 456, and Bull. Soc. Ent. Ital. xxxix. 1908, p. 106.

Arista with five hairs. Front moderately broad—one-fifth the breadth of the head. Tarsi reddish yellow.

Status uncertain: described from the female only.

Loc. Abyssinia.

4. Lyperosia punctigera, Austen.

Austen, Ann. Mag. Nat. Hist. (8) iii. 1909, p. 285; Bezzi, Archiv. de Parasitol. xv. 1911, p. 132.

This species, like *L. minuta*, Bezzi, has six hairs on the arista, but can be immediately recognised by the peculiar markings of the abdomen, which is olive-grey; the dorsum has a sepia-coloured median spot or tapering stripe on the second and following segments; the second and third segments in addition have each a pair of large and conspicuous transversely-oval clove-brown spots; the first posterior cell of the wing is narrowed at the tip and the posterior transverse vein is strongly bent outwards.

Loc. Uganda and the Nile.

5. Lyperosia exigua (Meijere).

Vide supra (under Asiatic species).

An Asiatic species which has been reported from Khartoum.

6. Lyperosia thirouxi, Roubaud.

Roubaud, C. R. Soc. Biol. Paris, 1906, p. 896.

Eyes of the male almost in contact; arista with six to eight hairs. *Proboscis short, swollen at the base*; first posterior cell hardly narrowed distally. Abdomen wholly black. Legs dark.

Bezzi considers that this species, having a short proboscis, would be more properly placed in the genus *Stygeromyia*.

Loc. Senegal.

D.—AMERICAN SPECIES.

1. Lyperosia irritans, L.

(*Vide supra.*)

This European species is found in N. America from Canada to Texas.

"Lyperosia" alcis, Snow.

(Vide supra *Hæmatobia alcis.*)

According to Bezzi this species should be included in the genus *Lyperosia*.

E.—AUSTRALIAN SPECIES.

1. Lyperosia exigua (Meijere).

(*Vide supra* under Asiatic species.)

Numerous specimens of both sexes of this species have lately been brought to the School by Dr. C. L. Strangman, who caught them on cattle in the neighbourhood of Port Darwin in the Northern Territory of Australia.

Stygeromyia, Austen.

Austen, Ann. Mag. Nat. Hist. (7) xix. 1907, p. 445; Bezzi, Zeitschr. f. Hymenopt. u. Dipt. vii. 1907, p. 416, and Archiv. de Parasitol. xv. 1911, p. 123; Brunetti, Rec. Ind. Mus. iv. 1910, p. 82; Alcock, Entomology for Medical Officers, 1911, p. 158.

"Intermediate between *Stomoxys*, Geoff., *Hæmatobia*, Rob.-Desv., and *Lyperosia*, Rond. (sensu Bezzi). General appearance and form of body like *Stomoxys*. Shape of proboscis and palpi like *Hæmatobia*. Arista feathered only on upper side, like *Stomoxys* and *Lyperosia*. Head somewhat flattened from front to rear: proboscis short, stout, shiny, not tapering, chitinous; palpi as long as the proboscis, clavate towards tips, curving upwards. Posterior cell of the wing narrowly open at the tip, and less than one-half the width of that cell-opening in *Stomoxys calcitrans*, L. Apical portion of the fourth vein beyond the bend perfectly straight, not bent inwards at extremity as in *Stomoxys* and *Hæmatobia*."

Geographical Distribution. Ethiopian Region (Africa and Southern Arabia).

1. Stygeromyia maculosa, Austen.

Austen, Ann. Mag. Nat. Hist. (7) xix. 1907, p. 447.

"Length $6\frac{1}{4}$ mm.; length of wing $6\frac{1}{2}$ mm.; width of head $2\frac{1}{3}$ mm.: width of front at narrowest part $\frac{1}{2}$ mm.: proboscis from base of chitinous part to the tip of labella $1\frac{1}{4}$ mm.: width of thorax at transverse suture $2\frac{1}{2}$ mm.: width of abdomen at hind margin of second segment $2\frac{3}{4}$ mm. Colour smoke-grey, striped and spotted with brown; bristles and hair entirely black; proboscis dark chestnut-brown; palpi and tibiæ orange-buff."

Thorax with a pair of narrow brown admedian stripes from anterior margin across anterior suture. A median stripe lighter brown begins just in front of inner dorso-central bristles, disappears close to hind margin, and reappears as a faintly marked fleck on base of scutellum. First segment of abdomen unspotted, second, third, and fourth with an elongated median spot of mummy-brown and a lateral spot on each side. Median spots on the second and third segments are guttate, that on the second segment is in contact with front margin but does not quite reach the hind margin; on third segment it is not in connection with either margin; on the fourth segment the median spot is a narrow median stripe from the front margin to a point one-third of the length of the segment from the hind margin. Legs greyish except the tip, which is orange-buff.

Loc. Aden.

2. STYGEROMYIA SANGUINARIA, Austen.

Austen, Ann. Mag. Nat. Hist. (8) iii. 1909, p. 286.

"Length, ♂ 7 mm., ♀ 6·75 mm.: width of head, ♂ 2·6 mm., ♀ 2·4 mm.; width of front at vertex, ♂ 0·5 mm., ♀ 0·6 mm.; length of wing 7 mm. Grey, clothed with black hairs and bristles, ♀ somewhat darker than ♂; closely resembling *S. maculosa*, Austen, in general appearance and also in markings of dorsum of thorax, but distinguished by the abdomen being without sharply defined mummy-brown spots, not in contact with hind margins of the segments, and by the proboscis, except tip, being orange-buff or ochraceous-buff, instead of dark chestnut-brown; dorsum of abdomen with a narrow, dark brown, median, longitudinal stripe, and more or less distinct dark blotches on hind borders of the second and third segments."

3. STYGEROMYIA WOOSNAMI, Austen.

Austen, Bull. of Entomol. Research, iii. pt. 1, May 1912, p. 97.

"Distinguished from both *S. maculosa*, Austen, and *S. sanguinaria*, Austen, by its much larger size; further distinguished from *S. maculosa* by its more strongly marked thorax and very different abdominal markings, and from *S. sanguinaria* by the frontal stripe being much darker (clove-brown instead of walnut-brown) and distinctly wider below (instead of, as in the male of *S. sanguinaria*, of practically uniform width throughout), by the proboscis being clove-brown instead of orange-buff or ochraceous buff, and by the femora being strongly suffused with dark grey instead of mainly buff or ochraceous buff."

Loc. East Africa Protectorate, 7500 feet.

? STYGEROMYIA THIROUXI, Roubaud.

Lyperosia thirouxi, Roubaud, *vide supra*.

According to Bezzi (Archiv. de Parasitol. xv. 1911, p. 126) this is probably a *Stygeromyia*.

Loc. Senegal.

PHILÆMATOMYIA, Austen.

Philæmatomyia, Austen, Ann. Mag. Nat. Hist. (8) iii. 1909, p. 295; Brunetti, Rec. Ind. Mus. iv. 1910, p. 89; Alcock, Entomology for Medical Officers, 1911, p. 159.
Pristirhynchomyia, Brunetti, Rec. Ind. Mus. iv. 1910, p. 91.

"Greyish flies, not unlike *Musca domestica*, L., in general appearance, but distinguishable at once owing to the remarkable proboscis. Front in male narrow, its width in centre being from one-eleventh to one-fifteenth of total width of head; width of front in female at vertex one-third of total width of head; proximal portion of proboscis (mentum) a strongly swollen chitinous bulb, distal portion soft and fleshy, folded back under distal end of bulb when not in use, but when in use extended, its terminal section consisting of a 'tubular extension,' which is protruded from between the labella, and is surrounded at the distal extremity with a circlet of stout chitinous teeth; venation generally as in *Musca domestica*."

These flies are very like house-flies—the resemblance being emphasized by the retractile proboscis, which is not chitinous in all its extent, an excellent figure of which is given in Alcock's 'Entomology for Medical Officers,' p. 162.

Geographical Distribution. India, Ceylon, Sokotra, Cyprus, Senegal, and the Congo Free State.

1. PHILÆMATOMYIA INSIGNIS, Austen.

Austen, Ann. Mag. Nat. Hist. (8) iii. 1909, p. 298, and African Blood-sucking Flies, 1909, pp. 137, 138; Brunetti, Rec. Ind. Mus. iv. 1910, p. 90.

"Length, ♂ 3–5·8 mm., ♀ 4–6½ mm.; width of head, ♂ 1·3–2·2 mm., ♀ 1·4–2·25 mm.; width of front in centre, ♂ 0·08–0·2 mm., ♀ 0·06–1 mm.; length of wing, ♂ 3–5·5 mm., ♀ 3·2–5·6 mm. Colour smoke-grey or yellowish grey: dorsum of thorax with four dark longitudinal stripes, broader and clove-brown or blackish in ♂, narrower and dark grey or brownish in ♀: dorsum of abdomen with a transversely elongate dark blotch on first segment, not reaching hind margin, and a small clove-brown median triangle at base of second and third segments, sometimes with a small mark at the base of the fourth segment; bristles black; palpi ochraceous-buff; wings hyaline or with slight ochraceous tinge; legs black; femora greyish."

Geographical Distribution. As above.

According to Patton and Cragg (Ann. Trop. Med. & Parasit. vol. v. no. 4, 1912) the female lays fifty to sixty eggs in fresh cowdung, and the larva, which is bright lemon-yellow in colour, passes through its post-embryonic development very quickly.

MISCELLANEOUS NOTES FROM THE ENTOMOLOGICAL DEPARTMENT.

By A. ALCOCK.

1. FEMALE DIMORPHISM IN *PHLEBOTOMUS MINUTUS*, Rondani.—A large number of specimens of a *Phlebotomus* were sent to the School from Sierra Leone by our good friend Dr. H. E. Arbuckle, who stated that the consignment was composed of two species, which were easily distinguishable in life. On sorting also there appeared to be two species—a lighter species with narrower wings and a darker species with broader wings. On mounting and examining the specimens, however (about a hundred were thus treated), they all proved to be Rondani's species *Phlebotomus minutus.* The darker forms with broader wings were *all* females, while of the lighter forms with narrower wings some were male and some female. All the specimens were cleared and mounted in Canada Balsam, some of them being first stained with borax carmine; and a series of *camera lucida* drawings was made to facilitate comparison.

2. VARIATION IN THE SCALES OF THE SCUTUM OF *ANOPHELES* ("*PYRETOPHORUS*") *COSTALIS*, Loew.—*Anopheles costalis*, known in medical literature as "*Pyretophorus*" *costalis*, is well understood to be a variable species as regards colouring, but the examination of a very large number of specimens

from West Africa, that have been recently sent to the School, shows that the form of the scales of the scutum is also subject to a good deal of variation.

These scales are usually described as "narrow curved," but the point which I wish to emphasize is that they often are considerably broader in the female than they are in the male.

In a paper on the Classification of the *Culicidæ,* published in the 'Annals and Magazine of Natural History' for August 1911, in which I have disputed the propriety of splitting the genus *Anopheles*, I have noticed that *Anopheles costalis* is to some extent intermediate between those species of *Anopheles* that have narrow scales on the scutum and those species that have broad scales on the scutum, and that certain specimens of *Anopheles costalis* to some extent resemble the species of the subgenus *Nyssorhynchus.* Recent observations confirm this opinion and strengthen the argument against splitting the genus *Anopheles.* [From the practical standpoint *Anopheles costalis* ♀ can always be distinguished from any species of the subgenus *Nyssorhynchus* by its slender palps.]

As I do not know the circumstances of the capture of the specimens from West Africa now under consideration, it is not possible to propose any explanation of the fact that the scales of the scutum in the females are sometimes broadish and sometimes quite narrow—whether or not it may be a seasonal variation.

3. A SUGGESTION FOR DESTROYING *CYCLOPS* IN SMALL COLLECTIONS OF WATER.—It seems worth recording that *Cyclops* is very sensitive to caustic potash; a solution of 0·15 per cent. kills them instantaneously, and a solution of 0·07 per cent. is fatal in 2 to 10 minutes. Occasionally a large egg-laden female still shows signs of life, in the weaker solution, at the end of 10 minutes. At least, this is the result of several experiments with a common British species.

These observations might find a practical application in villages where guinea-worm exists and the wells are known to be infested with *Cyclops*.

The weaker solution, which is fatal in a few minutes, works out at 1 lb. of caustic potash to about 180 gallons of water.

The cost of 1 lb. of caustic potash is about fourteen pence; and a solution of the strength of ·07 per cent., or about 7 grains to the pint, would not do any harm to men or domestic animals if a certain amount of it were drunk.

4. TWO NEW SPECIES OF BLOOD-SUCKING MAGGOTS DESCRIBED BY ROUBAUD.—In 'Comptes Rendus,' cliii. 1911, p. 553, Roubaud describes from the French Sudan a new genus of *Muscidæ*, the larvæ of which, like those of *Auchmeromyia*, suck blood. The animals attacked, so far as is known at present, are African Wart-hogs (*Phacochœrus*) and African Ant-eaters of the genus *Orycteropus.*

The new genus *Chœromyia* differs from *Auchmeromyia* in the form of the abdomen, which is squat-oval, with the 2nd, 3rd, and 4th segments subequal, the 4th segment, in the female only, being a little longer than the 2nd and 3rd.

Two species are described, namely, *Chœromyia boneti*, Roubaud, and *Chœromyia chœrophaga*, Roubaud.

[Journ. Lond. School Trop. Med., Vol. I. Pt. III., 1912.

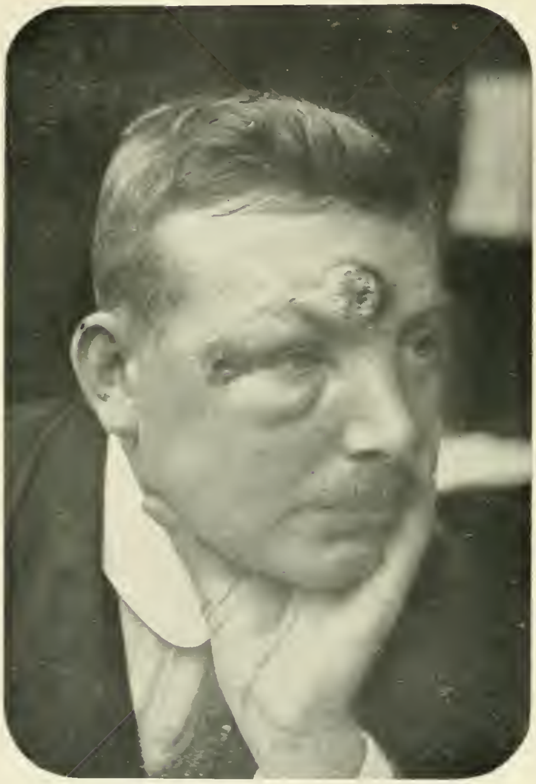

Photo by R. McKay.

A CASE OF LEISHMANIASIS FROM S. AMERICA.

II.—DEPARTMENT OF PROTOZOOLOGY.

A case of Dermal Leishmaniasis from S. America, with some Remarks on the Structure of the Parasite and its Culture.

By C. M. WENYON, M.B., B.S., B.Sc.,
Protozoologist to the London School of Tropical Medicine.

(With one Plate.)

The number of recorded instances of this disease from S. America are sufficiently few to justify the description of the present case, which was admitted to the Branch Hospital of the Seamen's Hospital Society at the Royal Albert Dock, under the care of Sir Patrick Manson, to whom I am indebted for the opportunity to investigate the case and to publish the following note.

The presence of leishmania in skin lesions in S. America was first demonstrated by Lindenberg, Carini, and Paranhos in 1909 in Brazil. In this year also a form of dermal leishmaniasis was shown by Tonin, Heckenroth, and Nattan-Larrier to occur in French Guiana, where it is known as Pian-Bois. In 1910 other cases were described by Matta and Rao on the Upper Amazon, and by Darling in Panama. Werner (1911) described a case in Rio de Janeiro; and Carini and Splendore have shown that there exists in Brazil a form of leishmaniasis which may affect the mouth and nasal cavities. Flu has described other cases from the three Guianas, and Escomel has lately described a disease known as espundia, which occurs in Peru and Bolivia. In smears from mouth and skin lesions obtained from Escomel, Laveran and Nattan-Larrier have found leishmania, and they describe a feature in the structure of the parasite which they think may indicate that it is a distinct species. This matter will be referred to again below.

The present case is that of an Englishman who travelled through Peru and Bolivia, from Lake Titicaca to the Tambopata river, and thence to its junction with the Rio Madre de Dios. He entered the country in July 1911, and the first indication of a sore was a small red spot over the right eye which appeared at the beginning of January 1912. On the way home this increased in size and an attempt was made to excise it, but with no good result. Later on another sore developed near the outer corner of the right eye.

Description of the Case.

The patient was first seen at the beginning of March. A photograph taken at this time is reproduced on the accompanying plate. He then had a large sore about three centimetres in diameter and situated over the right eye and root of the nose, with its lower limit in the eyebrow. It was in the form of a flat-topped tumour raised about half a centimetre above the surrounding skin. The surface was covered with granulations and fissures which showed a tendency to become scabbed over. On removal of the scab there escaped blood and pus which occupied the fissures between the granulations. The margin was covered with red œdematous skin which shelved down rather suddenly to the normal level. The surrounding skin was injected and inflamed for some distance around the sore. The second sore, at the outer corner of the right eye, was of a different character. Instead of being a raised tumour it was a punched-out ulcer measuring about 1 × ·5 centimetre. It was discharging a yellowish fluid. It may be stated that leishmania were found in both these sores. In addition to the two sores there was some involvement of the lymphatic system. There were two hard nodules below the skin of the eyebrow. These were not adherent to the structures below but moved with the skin. They were not painful on pressure. Running from the inner corner of the right eye across the cheek to a point just in front of the angle of the jaw was a hard cord, which was probably a much thickened lymphatic vessel. The lymphatic glands below the jaw were enlarged and formed a hard mass which was distinctly visible. Another and similarly thickened lymphatic vessel ran from the outer end of the right eyebrow across the face to the pre-auricular glands. There was considerable œdema of the upper and lower eyelids on the right side. No attempt was made to discover if leishmania were present in the lymphatic glands, so it is impossible to say whether this glandular enlargement was due merely to septic absorption or to the specific parasite. There was very little, if any, pain accompanying these various lesions, nor were there any noticeable constitutional symptoms. On one occasion while travelling in Peru, where the disease was contracted, the patient suffered from fever, which was relieved by quinine.

In character the sores themselves show no differences from those which I have seen in Bagdad and other parts of Asiatic Turkey. Both the raised and punched-out types occur in the East, and they present exactly the same features as in this case, with the exception of the somewhat extensive involvement of the lymphatic system, which I have not seen before. In this connection it is interesting to note that Werner has described a somewhat similar condition of the glands in a case seen by him in Rio de Janeiro. In Werner's cases parasites were found in distant lymphatic glands.

The disease described by Escomel from Peru and Bolivia, under the name of espundia, commences with a primary lesion in the skin. This heals, but eventually, it may be after several years, lesions appear in the mouth and nose and produce a profound cachexia from which the patient may die. It is in cases of this disease that Laveran and Nattan-Larrier have discovered the leishmania. It is impossible to say whether the present case is one of espundia or not. If so the two sores must be regarded as the primary lesions—the "chancre espundique" of Escomel.

Description of the Parasite and some remarks on a supposed peculiarity in the S. American Leishmania.

I was successful in finding leishmania in both sores. Material was obtained by removing tissue through the unulcerated margin of the sores. The discharge from the sores themselves contained so many bacteria and so much pus that, if leishmania were present they were obscured from view.

In size and shape the parasites corresponded exactly with those that I have seen in the sores of Asiatic Turkey. Typical oval or oat-shaped parasites showing both the rounded and large trophonucleus and the deeply staining dot or rod-like kinetonucleus were seen. In addition to the typical parasites there occurred many elongated and peculiarly shaped forms. These I have figured in my report on the Eastern Sore[*], and I have pointed out that in the dermal lesions the variety in the shape of the leishmania is much greater than in the case of those obtained from cases of Kala Azar. This fact would enable one to recognise a smear of Oriental Sore from one from a case of Kala Azar. The same can be said of the parasites obtained from the case of the S. American sore now under consideration.

As I have already mentioned, Laveran and Nattan-Larrier were the first to describe leishmania from the disease in Peru and Bolivia. These observers noted a feature in the parasites which they thought might be of specific importance. In all the films examined (a series of films sent to Paris by Escomel) the authors noted that the trophonucleus, instead of being rounded and separated from the margin of the leishmania, was very much flattened out against the fine limiting membrane of the parasite. In many instances it was reduced to a thin layer of chromatin lying along one side of the parasite, and often extending round one end. They assert that in their preparations this appearance is shown by all the parasites, while an examination of films of the Eastern Sore has failed to reveal this peculiarity. They suggest on this account that the S. American leishmania may be a distinct species. In films made from the present case I have found many of the forms described by these observers, but, in addition, there occur (both in and out of the cells) the usual typical leishmania without any nuclear flattening: moreover, between these and the typical forms all intermediate stages can be traced. Further, on looking through films made in Bagdad, I find that the same type of parasite exists there also. In one film the appearances were especially striking, for in one part of the film all the parasites showed this flattening of the nucleus, while in other parts they were normal. I cannot avoid the conviction that this flattening of the nucleus is the result of some change undergone by the parasite in the drying process of the film. In some instances it may be a feature of some degeneration while still in the tissues, but there results this important fact that the peculiarity described by Laveran and Nattan-Larrier occurs both in films made from sores of the East as well as in those from S. America, so that this feature cannot be held to indicate a difference in the species of leishmania.

Culture of the Parasite.

This was done in the now well-known N. N. N. medium. The skin over the margin of the large sore on the patient's forehead was carefully cleaned with acetone and ether, and finally painted with alcoholic iodine solution. By means of a fine glass pipette forced through the skin material was obtained for inoculating the tubes. In all, six tubes were thus inoculated, and it is interesting to note that though the sore was discharging a very foul material containing much pus and innumerable bacteria, an uncontaminated culture of flagellates resulted in every tube. This fact speaks well for the sterilising effect of the cleansing methods adopted. In all cases flagellates were first seen on about the tenth day. The cultures increased in richness, large clumps of organisms appeared, and by subculture—now at the ninth generation—the culture has been maintained for over two months. The organism grows readily both in the condensation water in this medium as well as on the surface of the agar. In the latter situation the flagellates tend to be plumper and rounder than when they are growing in the liquid medium. Comparing these cultures with those of the parasite of the Eastern Sore now obtained by many observers, including the present writer, I cannot find that any differences exist between the two. There is a tendency to the production of two types of flagellates. One is pear-shaped, with little difference in its length and breadth, and with the flagellum springing from its blunt end. The other form is elongated, with, however, the end bearing the flagellum more obtuse than the other. In cultures every intermediate form between these two exist, as also every shape of non-flagellated organism. All these forms were found in cultures obtained by me from the Bagdad Sores. It appears, however, that certain differences between the cultures of the *Leishmania tropica* and those from cases of Kala Azar do exist. Dr. Row has pointed out these differences, and I am at present engaged in testing this point.

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[*] "Oriental Sore in Bagdad, &c." Parasitology, October 24th, 1911.

Treatment of the Sore.

The case under consideration has been treated in two ways. The small sore at the outer corner of the eye received daily applications of the ointment recommended by Cardamatis and Melissidis, and consisting of equal parts of methylene blue, lanoline, and vaseline. At the same time the patient was given subcutaneous injections of killed cultures according to Dr. Row's method. On the first occasion (March 30) the patient received $\frac{3}{8}$ c.cm. of rich culture to which had been added forty minutes before five drops of glycerine. This had the immediate effect of rendering the flagellates immobile, and after the expiry of forty minutes it was concluded that they were dead. Some time after a single moving flagellum was seen in some of the fluid remaining, so that the parasites could not have been all killed at the time of injection. On April 9 a second injection of ·5 c.cm. of culture mixed with ·5 c.cm. of glycerine an hour before were given. On neither occasion was there any constitutional disturbance following these doses. The glycerine mixture was inoculated on each occasion on to blood agar tubes, but no growth occurred, so that though a motile organism was seen on the first occasion the parasites in the mixture were too much altered to grow.

After the second injection the small sore commenced to heal, though it had been showing a tendency to spread towards the eye, and a month later it had practically closed over with skin. This sore was being treated at the time with the ointment, so it is impossible to say which was the curative agent. The methylene blue ointment was then applied to the large sore, which has commenced to improve. It has become less prominent, having now been reduced to the level of the surrounding skin. The margin is not œdematous or raised, and there is little inflammation. On two occasions since the commencement of the treatment I have failed to find parasites in the material obtained from the sore. The case is still under observation and will form the subject of a future report if the results justify this.

Successful Inoculation of a Dog.

With material obtained from the sore a small baboon * was inoculated in the skin of the nose and the tips of the ears. A dog was similarly inoculated in two places on the nose and on each ear. Though three months have elapsed since the inoculation the monkey has shown no signs of infection. The dog, however, has developed a sore on each ear, and in these leishmania have been found. The incubation was two months.

SOME CRITICAL REMARKS ON CAPT. PATTON'S REPORT ON ORIENTAL SORE.

By C. M. WENYON.

[Preliminary Report on an Investigation into the Etiology of Oriental Sore in Cambay, India. Capt. W. S. PATTON, M.B., I.M.S.—Scient. Mems. by Officers of the Med. and San. Dept., Gov. of India. No. 50 (New Series).]

IN this Report the author gives an account of observations made by him in Cambay, India, during the summer of 1910. Many of the observations correspond with those made by the present writer in Bagdad during the same summer, while in other respects the results differ.

Capt. Patton had under observation for three and a half months a case in a boy ten years of age who had a sore of the non-ulcerating type on the lower part of the left thigh. On this case numerous experiments were conducted. It was noticed that the sore occasionally appeared more swollen than usual, and that the parasitic area as determined by puncture of the margin had extended. Parasites were then found not only in the macrophages but also in mononuclear and polynuclear cells. On the first occasion in which this phenomenon was observed two parasites were found in a large mononuclear leucocyte in a drop of blood taken from the upper third of the same thigh. On three other occasions parasites were found in the surface blood taken a considerable distance from the lesion. The author does not state if the blood was taken from the same leg on every occasion. Presumably it was. The observation is an interesting confirmation of Neumann's discovery, made in 1909, of parasites in the peripheral blood in this disease. The present writer made many attempts in Bagdad to discover parasites in the circulating blood both by direct examination and by culture methods, but without success.

Capt. Patton states that the parasites disappear quickly from the sore after it has broken down, and he quotes the history of a sore from which he suffered in support of this contention. He disagrees with Dr. Row, who has stated that the parasites persist in the sore to the very end. The present writer in Bagdad has found that the parasites are much more numerous and easily found in the non-ulcerating sores, but, as Dr. Row has said, parasites persist even in the ulcerating sores up to or at any rate nearly up to the healing stage. Often a very careful search is necessary, and unless tissue is removed from the base of the ulcer, or preferably through an incision in the non-ulcerating part of the margin, the parasites may not be found. Sometimes, however, the ulcerating sore discharged a fluid not only teeming with bacteria of many kinds but also containing innumerable leishmania.

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* Since writing the above I find the baboon (*Cynocephalus*) has shown signs of infection in the form of small papules on each ear and the nose at the sites of inoculation. Leishmania have been found in the papules on the ears. In this case the incubation has been a few days under four months. I find that in the last number of the 'Bulletin de la Société de Pathologie Exotique,' Splendore records the successful inoculation of monkeys with the virus of the dermal leishmaniasis of Brazil.

Capt. Patton criticises the view that the house-fly may be responsible for the transmission of the disease, and for the period of one month he daily attempted to infect scratches on his hand by allowing flies to suck up the parasite-containing exudation from a sore and then to feed upon abrasions on his hand. No sore developed at these places; so he thinks the fly is excluded as a carrier of the disease. He also points out that the people of Cambay are confident that the sore never commences in an abrasion, scratch, or open cut in the skin. Attempts were also made without success to produce a sore by placing some of the discharge containing parasites on the granulations of already existing sores of another kind. It is quite true that most usually the sore commences in healthy skin. Even if a cut or scratch were infected by the transference of a few parasites, the sore would only appear later after the wound had healed. This is the experience of all those who have practised direct inoculation by scarification; but to state that such wounds or sores never become infected requires further proof. I have the history of two Europeans who both contracted sores on the top of an abrasion. In one case an abrasion over the right cheek-bone resulting from a fall from a horse became infected, while in the other case an abrasion over the right shin-bone similarly developed into Oriental Sore.

Capt. Patton's feeding experiments with house-flies led to results similar to those obtained previously by Dr. Row and the present writer. The parasites ingested could only be recovered up to six hours after feeding, after which they quickly degenerated and disappeared. There was no indication of a development into flagellate forms. Dr. Row has suggested that the house-fly may take up parasites and later pass them, still in a living condition, in the fæces, and that sores may be infected in this way. There is nothing against such a possibility, though it cannot be accepted till experimentally proved. Alvarez has suggested that fleas may infect dogs with Kala Azar by passing living leishmania in their fæces, and quite recently Nöller has shown that forms of *Trypanosoma lewisi* passed in the fæces by fleas are infective for rats, which naturally become infected with this trypanosome by licking up the forms deposited by the fleas on their skin. Capt. Patton, however, says that in order for the transmission to take place the fly must have a clean alimentary tract. Why this should be necessary is difficult to understand, for certainly the leishmania may live and develop to some extent in association with bacteria. Against the fly hypothesis the author quotes the fact that judging from people who become infected after only a short stay in Cambay, the sores are most commonly contracted, not in the fly season but in the cold weather. In Bagdad, however, I found that though most sores make their first appearance at the beginning of the cool season, indicating that infection has probably taken place during the hot weather, they may appear at any time of the year.

Experiments conducted with *Pediculus vestimenti* and *Pediculus capitis* gave negative results.

With *Stegomyia* sp. Capt. Patton conducted experiments; but though these fed upon the sore, no parasites were discovered in them. With *Stegomyia*

I was more fortunate in Bagdad, for there the mosquito readily took up parasites from the sore and in a certain number of instances these developed into flagellates in the gut of the insect.

No evidence could be obtained as to dogs suffering from the disease in Cambay.

With bed-bugs the results obtained were similar to those obtained previously by me in Bagdad ("Oriental Sore in Bagdad," Parasitology, Oct. 1911). It was found that the parasites taken up by the bugs developed into flagellate forms. The author makes the statement that the parasites ingested by the bug when feeding from the margin of the sore cannot possibly have been dislodged from the sore, but have come from the blood circulating at the spot. The proboscis of the bug is not long enough to reach the parasites. This statement is doubtful, for I have examined sections of excised sores in which the layer of epidermis separating the large macrophages full of parasites from the surface is exceedingly thin, not more than a fraction of a millimetre (about 50 microns) in thickness. It would certainly be possible for bugs to take up parasites through such a layer. Further, the author states that bugs will not feed from the surface of a sore-discharging parasite. In Bagdad I found that bugs would feed fairly readily in this way.

In order to obtain development of the leishmania into flagellate forms Capt. Patton found it necessary to keep the bugs at a suitable temperature, 22°–25° C. He found that at the low temperature of 18°–20° C. the blood in the gut of the bug, and especially of the nymph, remained undigested for a period of 48 hours, and so keeps in good condition for the development of the leishmania. He points out that Oriental Sore only occurs in those parts of India where there is a decided cold season, as, for instance, Karachi, Quetta, Jacobabad, Dera Ismail Khan, Delhi, Lahore, Cambay. As a result he cannot help thinking that there is a factor of fundamental importance underlying the restricted geographical distribution of Oriental Sore in India, and he is convinced that the explanation is to be found in the relation of the temperature to the digestive processes in the bug. In adult bugs only a partial development took place, and the infection was found to die out in about ten days. In the case of the nymphs, however, a much more complete development occurred, and in one instance the author thinks he has seen the post-flagellate stage of the leishmania. It is interesting to note that many of the bugs were fed at a distance from the sores, so that they could only ingest parasites in the circulating blood. On one occasion 100 of such bugs were dissected, and in four of these parasites were found—some in unchanged condition still in leucocytes, and all the intermediate stages showing the formation and extrusion of the flagellum and the multiplication of the resulting flagellates up to the long mature forms. The dissections were made at intervals of one to five days after feeding.

Many attempts were made to transmit the disease by feeding bugs upon cases of the disease in Cambay and afterwards on susceptible persons in Bombay, but without result. Capt. Patton also attempted to transmit the disease from himself to a susceptible person by means of nymphs of the bed-bug, but also with a negative result. From the results obtained he concludes that the nymph of the bed-bug (*Cimex rotundatus*) is the transmitter of the Cambay Sore. He makes many statements in support of this. Firstly, that 95 per cent. of the sores in Cambay are contracted in the cold weather. Secondly, that the bed-bug is the only blood-sucker which will when disturbed commence feeding again a short distance from its first puncture. This will explain cases of multiple infection. Thirdly, the author states that an insect such as the bug, which is constantly present and returns to suck the blood of the same patient on many consecutive nights, is more likely to take up parasites which only periodically occur in the blood, than an insect such as the mosquito, which hardly ever sucks the blood of the same person on more than two or three occasions. It is difficult to understand the meaning of this statement. The more often any individual insect feeds the more likely it is to take up parasites, but it matters not whether the insect is one that has previously fed on the case or not, so long as it is feeding at the right time, and this must happen as well for the previously fed bed-bug as for the mosquito at its first feed. Fourthly, the author records the histories of two cases in Europeans where the Oriental Sores developed at spots upon which bed-bugs had previously fed. Histories of this nature are interesting, but the present writer collected several such, and it appears that they are hardly reliable though given in all good faith. In the case of three Europeans who developed Oriental Sores in Bagdad or Aleppo a definite history of a mosquito bite (in which the mosquito was actually seen) becoming a typical sore was obtained at first hand from the victims themselves. In another instance a similar history relating to the sand-fly (*Phlebotomus*) was obtained. and in this case leishmania were found in the resulting sore. The author finds that the limitation of the disease to large towns can only be explained by the bed-bug hypothesis, and further that the occurrence of sores almost entirely on exposed surfaces of the body supports his theory, for in his opinion the bed-bug feeds from such surfaces in preference to any covered part of the body.

The author inoculated himself from a sore and developed a typical lesion in which leishmania were found after the short incubation of sixteen days. In another instance a monkey developed a sore in eighteen days; while in another monkey the incubation was two months. This has led the author to the conclusion that the incubation varies with the number of parasites injected.

In conclusion Capt. Patton states that he has no hesitation in saying that the house-flies *Musca nebulo* and *Musca* sp. play no part in the transmission of the disease in Cambay, and that he has no doubt whatever that the bed-bug *Cimex rotundatus* is the only insect transmitter of the disease in Cambay. This may be true of Cambay and other parts of India, but it certainly is not true of Bagdad, as has been pointed out by the present writer in his report on Oriental Sore in this district. However, we must await the conclusive transmission experiments before a definite answer can be given to these questions, and it seems to the present writer that the fact that development into flagellates occurs in the gut of an insect is not sufficient proof of this insect's ability to transmit the disease. Many of the results recorded in this interesting paper agree with those obtained by the writer in Bagdad, whose report Capt. Patton had evidently not seen when he wrote up his results.

HARTMANN AND WITHMORE ON THE PARASITIC AMŒBÆ OF MAN.

[Untersuchungen über parasitische Amöben:—

I. HARTMANN, M.—*Entamœba histolytica* Schaudinn. Archiv für Protistenkunde, Bd. xviii.
II. HARTMANN, M.—*Entamœba tetragena* Viereck. Ibid. Bd. xxiv.
III. HARTMANN, M., and WITHMORE, E. *Entamœba coli* Lösch em. Schaudinn. Ibid. Bd. xxiv.]

IN the first of these papers the author gives an account of *Entamœba histolytica* in confirmation of much of Schaudinn's description of this pathogenic amœba. The paper was published in 1909, and since that time the author has been able to study a greater amount of material and also to examine many of Schaudinn's original preparations. The result of this further study is embodied in the second of the above papers. In it the author retracts much of what he stated before, and concludes that both Schaudinn and he were in reality working with the amœba now known under the name of *Entamœba tetragena*, and that paucity of material led to errors of description in the life-history of this amœba, so much so that when the true life-history of the pathogenic amœba was discovered it differed so much from the original erroneous description that Viereck thought it to be a distinct species and gave it the name of *Entamœba tetragena*. If this be true, then the terms *Entamœba histolytica* and *Entamœba tetragena* are synonymous. It comes about, however, that the amœba which is the commonest cause of dysentery in many countries (Africa, Congo, Cameroons, Togo, West Africa, Egypt, South America, Central America, India, Java, Sumatra, Philippines, Formosa, China) answers in its structure and cyst-formation, not to Schaudinn's *Entamœba histolytica* but to Viereck's *Entamœba tetragena*.

In the present study of *Entamœba tetragena* the author has obtained material from various parts of the world and also from experimentally infected cats, in which it has been only possible to carry on the infection through two passages, as this animal is not a good host for the amœba, which quickly degenerates and changes in this unnatural environment.

The amœba is large, measuring 25–40 μ in diameter. Smaller forms (20 μ) occur prior to encystment, while the smallest individuals observed measure only 5–10 μ. The movements and structure of the amœba correspond closely with Schaudinn's and Jurgens's descriptions of *Entamœba histolytica*, for these observers were in reality working with *E. tetragena*. There is the homogeneous highly refractile tough ectoplasm surrounding the granular less refractile endoplasm, a character which has been supposed to enable one to separate the *E. coli*, which does not show such a marked distinction of ecto- and endoplasm. The author has found, however, that these are not constant features and that *E. tetragena* may on many occasions have no distinct ectoplasm, so that it comes to resemble *E. coli*, with the result that in order to distinguish the two it is necessary to pay attention to other points, viz. nuclear condition, reproduction, &c. The present writer came to the same conclusion some years ago, and drew attention to the fact that it was not possible to certainly differentiate the pathogenic and harmless amœba by their morphology alone, as this was variable, but that it could only be done by a study of their life-histories ('British Med. Journal,' October 24th, 1908). In *E. coli* it is the rule to find a vacuolated protoplasm containing bacteria, a feature not seen in *E. tetragena*, in the endoplasm of which red corpuscles are often found.

The nucleus of *E. tetragena* is clearly visible and resembles that of *E. coli.* There is a definite nuclear membrane on which chromatin granules are distributed. At the centre is a karyosome surrounded by a clear area. Between the clear area and the nuclear membrane is the linin network of the nucleus, upon which also chromatin is arranged. At the centre of the karyosome is a minute granule, the centriole. The nucleus is in constant activity, and it is found that in the living amœba the chromatin breaks away from the karyosome and passes out towards the periphery of the nucleus. This may go on till there is little but the centriole left at the centre of the nucleus. The centriole then increases in size by the deposit round it of more chromatin till the karyosome is reformed. The outward migration of chromatin then commences again. This cyclic change in the nucleus is very readily seen in *E. tetragena*, though it occurs to a less extent in *E. coli* and in fact in many other Protozoa. It may be used as an aid to differentiation in the two species.

The only method of multiplication is by simple division. No schizogony of *E. tetragena* has been observed. In nuclear division the centriole is the first object to divide. It becomes drawn out and constricted, and there results a modified mitosis.

The author continues that in dysenteric stools, and especially in the fæces of experimentally infected cats, very many of the amœbæ are degenerate and can easily lead one to wrong conclusions. It is to such forms that much of the confusion of descriptions is due, and many of the so-called cases of *E. histolytica* are of this nature, as also are many of the pathogenic amœbæ described as distinct species. Certain appearances in these degenerate amœbæ formerly led the author to think that a process of autogamy occurred in its life-history; he now finds that no such autogamy takes place.

The formation of cysts occurs very rarely in the case of *E. tetragena.* Before encystment the amœba becomes smaller and chromatin appears in the cytoplasm in the form of granules or rods, which run together into clumps three to six in number. The author has called the latter chromidial bodies, though he is not quite clear as to their nature. These chromidium-containing amœbæ occurred in Schaudinn's preparations, and he thought them to be his *E. histolytica* and distinct from the ordinary forms of *E. tetragena* occurring in the same preparations, which he thought were *E. coli* and represented a double infection with both forms.

The cyst is entirely filled with protoplasm, though at the centre there may be a large vacuole, which occurs, however, much less commonly than in *E. coli* cysts. Within the cyst nuclear division takes place much as in the division of the nuclei of vegetative forms. After this division the cyst contains four nuclei. Never have more than four been observed nor has any autogamy been seen. The author thinks that the cysts are taken up by a new host, that there the protoplasm divides into four, and that by rupture of the cyst four amœbæ escape. These are probably gametes which will conjugate in pairs, just as Mercier has shown to occur in the case of *E. blattarum* of the cockroach.

In the third of these papers Hartmann and Withmore give a detailed description of *Entamœba coli.*

In size there is little difference from *E. tetragena*, but usually there is no distinction between ecto- and endoplasm. Only rarely have the authors seen a red blood-corpuscle in the endoplasm, which in this amœba is generally much vacuolated and contains cocci and bacilli. The nucleus differs very little from that of *E. tetragena*, and the same cycle of changes takes place in the karyosome, but they are not so clearly defined. The authors describe one peculiarity in the nucleus of *E. coli*. This is in the karyosome of about half the individuals examined. Instead of being a single granule as in *E. tetragena*, it is composed of two chromatin granules connected by a bridge of achromatic substance. The authors think this point may be of diagnostic importance. Schaudinn described the nuclear division of *E. coli* as being amitotic, but the authors regard it as a modified mitosis. Schaudinn further described for *E. coli* a multiple division or schizogony. In this process the chromatin of the nucleus became clumped on the nuclear membrane in eight masses, which separating simultaneously gave rise to eight daughter nuclei. The authors cannot confirm this process of nuclear division, but regard the multinuclear forms as resulting from a repeated division into two, for they have seen amœbæ with two, four, and eight nuclei.

As regards the encystment of *E. coli*, the authors have come to the conclusion that the autogamic process described by Schaudinn as taking place within the cyst does not occur. Within the cyst the single nucleus by a thrice repeated mitotic division gives rise to eight daughter nuclei. Frequently after encystment a large quantity of chromidium appears in the protoplasm, and this eventually may be absorbed or run together into several masses, as occurs in the cysts of *E. tetragena*. A large vacuole frequently appears in the protoplasm, but when the eight nuclei stage is reached the vacuole has usually disappeared. The authors do not think that an autogamy takes place within the cyst, so they believe that as in the case of *E. tetragena* the eight amœbæ which eventually escape from the cyst in the next host are gametes, which are destined to conjugate in pairs before giving rise to the vegetative forms of *E. coli*. Occasionally there are encountered cysts containing ten, twelve, or even a larger number of nuclei. Such were observed by Schaudinn, and others have described them. They probably represent some abnormal development. C. M. W.

On the Cultures of Amœbæ obtained from the Intestine of Man.

[Aerial Contamination as a Fallacy in the Study of Amœbic Infections by Cultural Methods. Wells, R. T. Parasitology, iv. 1911.]

[*Amibe limax* (*Vahlkampfia*, n. gen.) dans l'intestin humain. Son importance pour l'interprétation des amibes de culture. Chatton et Lalung-Bonnaire. Bulletin Société de Pathologie Exotique, Feb. 1912.]

[Parasitäre und freilebende Amöben aus Manila und Saigon und ihre Beziehungen zur Dysenterie. Whitmore, R. Archiv für Protistenkunde, Bd. xxiii. 1911.]

Musgrave and Clegg (1904) cultivated amœbæ from the fæces of dysenteric patients in the Philippines and concluded that these amœbæ were identical with the entamœbæ from the intestine. In 1905 Lesage similarly cultivated an amœba from dysentery cases in Saigon and Toulon. It was assumed by these observers that the cultures were those of pathogenic amœbæ. In a study of these amœbæ in Manila and Saigon, Whitmore has come to the conclusion that the resulting cultures are not of either *Entamœba tetragena* or *coli*, the two parasitic amœbæ of man to be met with in these parts, but are cultures of amœba of the *limax* type, whose natural habitat is outside the human body. Shortly after the publication of Musgrave and Clegg's results I attempted to cultivate amœbæ from cases of dysentery in which these organisms were found. The technique of Musgrave and Clegg was carefully carried out, and as a result cultures of amœbæ were obtained. The amœbæ, however, differed so markedly from the true entamœbæ of the gut that it was concluded that the amœbæ cultivated were not those of the gut but probably free-living forms of the *limax* type, which had accidentally been present in the fæces either as a contamination from without or as encysted forms which had been ingested with the food. A number of experiments with the encysted forms obtained on agar plates showed that these were able to pass through the intestine of mice without losing their vitality. Similar results were obtained when attempts were made to cultivate entamœbæ of the mouse and guinea-pig. These results were published in 1907 (C. M. Wenyon, "Observations on the Protozoa in the Intestine of Mice," Arch. für Protistenkunde, Suppl. I.). Since that time many other observers have come to a similar conclusion (Hartmann, 1908 & 1909, Werner, 1908, Nägler, 1909). Wells has recently shown how easily material containing entamœbæ of the gut can be contaminated with amœbæ apparently floating about in the air in the encysted form.

In this connection the observations of Chatton and Lalung-Bonnaire are of much interest. A patient in Indo-China suffered from attacks of diarrhœa. In the fresh stool there were only found those bodies, measuring 10–15 μ in diameter, which have been held to be encysted forms of *Trichomonas*, but which Alexeieff has shown to be yeast-like cells to which he has given the name of *Blastocystis enterocœla*. In films fixed and stained by the wet method in addition to these bodies there were found a few amœbæ which were peculiar in that their nuclei differed from the typical nuclei of the entamœbæ. Instead of the chromatin being distributed in small granules over the nuclear membrane there was a large central karyosome as in the nucleus of *Amœba limax*. The amœba was further peculiar in that it apparently fed on one special bacterium, several chains of which were often found in the amœbæ. They varied in diameter from 10–20 μ. It is unfortunate that this amœba was not observed in the living condition. Fresh fæces had been inoculated on to four tubes of Musgrave and Clegg's amœba medium. Twelve days later all four tubes showed cultures of amœbæ which corresponded in every way with those seen in the stained preparations of the fæces, except for the fact that the amœbæ were feeding on mixed bacteria instead of the special one mentioned above. In the cultures the author was able to follow out the details of nuclear division and the cyst formation which are identical with those of another amœba cultivated by the author and Delanoë from various sources, and which is probably a usually free-living form, *Amœba punctata*. The question arises, is this amœba cultivated by the authors the same as the one seen by them in the stained fæces? The structure of the stained amœbæ of the fæces corresponds with that of the amœbæ on the culture plates, and, further, the fact that a culture resulted on all four tubes inoculated leads the author to conclude that the amœbæ are identical. Had the cysts of the amœbæ or forms with dividing nuclei occurred in the fæces the authors might have been able to compare these processes with those of the amœbæ on the plates.

The authors then point out that there is a general tendency to regard the supposed cultures of entamœbæ obtained by Musgrave and Clegg, Lesage, Walker, Noc, to be really cultures of free-living amœbæ which happen to be present in the fæces in encysted condition at the time the cultures were made Against this view there is the serious objection that Noc, Gauducheau, Mathis and Leger, Walker, Liston, and Martin have obtained cultures of amœbæ from liver-abscess pus containing entamœbæ. Some, at any rate, of these observers claim to have taken precautions to obviate any accidental contamination from without. Wells has shown how easily aerial contamination may occur, but the authors point out that if it is assumed that the cultures have arisen in this way the several observers are accused of faults of technique. The present writer has tried on three occasions without success to obtain cultures by inoculating plates of Musgrave and Clegg's medium with pus from liver-abscess containing living amœbæ. The authors regard their discovery of the amœba of the *limax* type in their case as affording an explanation of the cultures obtained from liver-abscess pus. It is difficult to imagine how an encysted amœba could find its way to the liver, but a free form such as they have observed in this case could as readily pass from the gut to a liver-abscess as one of the entamœbæ themselves, so that liver-abscess pus containing entamœbæ, even if drawn off with every precaution against contamination, might give rise to the usual cultures in which the amœbæ are of the *limax* type and quite different from the entamœbæ.

The authors conclude their paper by suggesting that the name *Vahlkampfia*, n. gen., be used as the generic title of all those amœbæ whose nuclei both in rest and in division correspond with that of the amœbæ hitherto known as *Amœba limax*. These are mostly free-living forms and include the amœba which is the subject of this paper, *Vahlkampfia punctata*. It is usually a free-living amœba occurring in vegetable infusions, but may occasionally occur as a free amœba in the human intestine, from which it is readily cultivated on a suitable medium. As regards the genus *Entamœba*, the authors point out that it was created by Leidy (1879) for the parasitic amœba of the cockroach, and in 1897 Casagrandi and Barbagallo applied it to the parasitic amœbæ of vertebrates. In 1910 Mercier gave a detailed account of the life-history of the *Entamœba blattarum* of the cockroach. It differs completely from that of the parasitic amœbæ of man, which as a result cannot be included in the same genus. Accordingly the author suggests as the generic title of the amœbæ of vertebrates the name *Löschia*, in honour of the author who first named the human *Amœba coli* in 1875. C. M. W.

HIDEYO NOGUCHI ON THE CULTIVATION OF SPIROCHÆTES FROM SYPHILIS AND OTHER SOURCES.

[A Method for the Pure Cultivation of Pathogenic *Treponema pallidum* (*Spirochæta pallida*). Journal of Experimental Medicine, Aug. 1, 1911.]

[The Direct Cultivation of *Treponema pallidum*, Pathogenic for the Monkey. Ibid., Jan. 1, 1912.]

[Cultural Studies on Mouth Spirochætes (*Treponema microdentium* and *macrodentium*). Ibid., Jan. 1, 1912.]

[Pure Cultivation of *Spirochæta refringens*. Ibid., May 1, 1912.]

In the first of these papers the author refers to Schereschewsky's culture of *Treponema pallida* by inserting a piece of infected tissue deep down into gelatinised horse serum. Later Mühlens and Hoffmann both used this method for obtaining first generations, and later succeeded in purifying the strain from other bacteria by employing a horse serum agar. These investigators were never able to reproduce lesions with the cultures, though the organisms exhibited the typical characteristics of *Tr. pallida*. However, Bruckner and Galasesco and Sowade reported having obtained syphilitic lesions in rabbits by the inoculation of young impure cultures which still contained the original syphilitic tissue.

The present author obtained culture of six different strains of *Tr. pallida* by first inoculating the disease into rabbits' testicles and cultivating from this source. The medium employed is serum water (sheep, horse, rabbit) to which a piece of sterile rabbit-tissue is added (kidney or testicle). Tubes 20 cm. high and 1·5 cm. wide are filled with 16 c.c. of serum water (1 part serum and 3 parts distilled water). The tubes are sterilised on three days at 100° C. for 15 minutes each day.

A small piece of fresh sterile tissue of the rabbit is then added, after which the tubes are incubated at 37° C. for two days to ensure their sterility. To each tube a layer of sterile paraffin oil is now added to shield from the air and evaporation. Cultivation is carried on under strict anærobic conditions by the combined use of hydrogen gas, vacuum, and pyrogallic acid in an anærobic apparatus. The medium should be slightly alkaline in reaction. The chief difficulty is in obtaining the primary culture: the subcultures grow under less strictly anærobic conditions. At first it was impossible to obtain a pure culture, as the *Tr. pallida* always grew with other organisms, but finally the author succeeded by permitting the *Tr. pallida* to grow through a Berkfeld filter. Later it was found that certain strains of *Tr. pallida* would grow with bacteria along the stab canal in serum agar tissue medium, but while the bacteria did not spread into the agar the *Tr. pallida* did, so that from the peripheral hazy part the author was able to obtain four pure strains of *Tr. pallida*. The growth is limited to the bottom of the tube in the neighbourhood of the piece of tissue.

In the serum water tissue *Tr. pallida* commences to multiply after forty-eight hours and continues to grow for at least four or five weeks. The morphology of *Tr. pallida* cultivated on solid media is quite typical, and it is impossible to distinguish it from specimens just taken from human or animal lesions.

Two of the pure strains of *Tr. pallida* when inoculated into the testicles of rabbits produced typical lesions in which enormous numbers of *Tr. pallida* occurred.

In the second paper the author has extended his observations and describes how he has successfully obtained cultures of *Tr. pallida* from human lesions. The maintenance of strict anærobic conditions is essential, and he expresses the belief that the piece of tissue in the medium acts as an oxygen abstracter. He employed high cylindrical layers of solid medium consisting of two parts of 2 per cent. slightly alkaline agar and one part of ascitic or hydrocele fluid at the bottom of which is a piece of sterile rabbit's kidney or testicle. This medium, when inoculated as a stab with fluid containing *Tr. pallida* and bacteria, seems to keep the latter fixed to the canal, while the *Tr. pallida* migrate to a distance and may often be found in pure or nearly pure culture. In preparing the medium after solidification a layer of melted paraffin wax is poured on to the surface of the agar. To inoculate the tubes pieces of chancre, condyloma, or skin papule previously cleansed with sterile salt solution are employed. These are immersed immediately in sterile salt solution containing 1 per cent. sodium citrate and cut up into fragments. One fragment is ground up with citrate solution in a mortar. Six tubes should be inoculated by forcing down to the bottom of the tube a piece of the tissue and then inoculating the canal thus produced with several drops of the emulsion. Care must be taken not to tear the medium. The tubes are incubated at 37° C. The medium becomes opalescent and shows a dense opaque growth of bacteria along the canal. Gas is produced and the medium torn. The medium is punctured in several places with a capillary pipette and examined with the dark ground. In the agar itself the *Tr. pallida* are not motile, but should a space exist they show active motility. Purification is begun by inoculating several tubes with the impure culture by means of a charged capillary pipette which is inserted quite to the bottom of the agar. Growth of bacteria again occurs along the track and *Tr. pallida* spread out into the agar. Subcultures are made from the peripheral part by cutting the tubes across, sterilising the surface of the agar with sublimate alcohol, removing the excess with sterile absorbent gauze, and causing a crack to occur in the agar column by bending it. From this fissure, which must not extend to the central canal, subcultures are made. Sometimes pure cultures of *Tr. pallida* are quite invisible, so that the dark ground microscope must be used to test its extension. When hydrocele or ascitic fluid is not available serum from the horse or sheep may be used for subcultures, though the former is better.

In young cultures the spirochætes are short and actively motile. After about two weeks they attain the usual length of eight to ten curves. The ends of the mature *Tr. pallida* often show fine flagellum-like prolongations which are frequently perfectly curved like the body.

As regards the method of division, the author recalls the fact that Schaudinn first described longitudinal division of the organism. Since then both longitudinal and transverse division have been described. In cultures all stages of longitudinal division can be seen, and these are readily followed on the dark ground microscope. The process is slow and takes about two hours for completion. The final result is two symmetrical halves, although asymmetrical division has been observed in rare instances.

Inoculation of pure culture into the skin of two species of lower monkeys resulted in the production of lesions resembling those of primary syphilis. During the development of the lesions the blood acquired the power of giving a positive Wassermann reaction. The monkeys were inoculated on the eyebrow and also on the scarified prepuce, scrotum, or labia.

In the third of the papers here reviewed the author has described cultures of mouth spirochætes. He points out that three kinds of mouth spirochætes have been recognised. One is larger and thicker than the others and has only a few flat curves; it is known as *Spirochæta buccalis* (Cohn, 1878). The smaller type is *Spirochæta dentium* (Koch, 1877), while the medium type has been called the medium form by Hoffmann and von Prowazek. Mühlens obtained cultures of spirochætes in which the organisms resemble to some extent *Sp. dentium*, but it is possible that he obtained mixed cultures of both the medium and small spirochætes. The author has now cultivated both these forms, and, as they are readily distinguished, proposes for them the names *Treponema microdentium* and *Treponema macrodentium*, for in general characteristics they agree with the *Treponema pallida* of syphilis. Cultures are obtained by inoculating sheep serum water containing sterile rabbit tissue, as in the case of *Treponema pallida*. Subcultures are made in agar media covered with paraffin oil as before, and in this way pure cultures can be obtained. The culture of *Tr. macrodentium* is more difficult and tedious than that of *Tr. microdentium*, and in order to obtain it, it is necessary to employ material in which the larger form predominates. The young forms of *Tr. macrodentium* have two to eight curves, may measure 0·7 to 0·1 micron in width and 3 to 8 microns in length. Older specimens have fourteen or more curves, measure 0·3 of micron in width and 12 or more microns in length. Both *Tr. micro-* and *macrodentium* may have a fine tapering flagellum at one or both ends, and they reproduce in culture, as *Tr. pallida*, by longitudinal divisions. Inoculated into rabbits they produce a temporary local induration, but nothing resembling the lesions produced by injection of *Tr. pallida*.

In the fourth of the papers here reviewed the author records the successful culture of *Spirochæta refringens*, the organism described in 1905 by Schaudinn and Hoffmann as occurring in lesions, both syphilitic and other, about the genitals. It is differentiated from *Tr. pallida* by its larger dimensions, its plump shape, its comparatively few irregular curves, and its readiness to take aniline dyes. Schaudinn and Hoffmann named it *Sp. refringens* and considered it to be non-pathogenic.

The author obtained a pure culture by employing his technique for the culture of *Tr. pallida*. It grows luxuriantly in the deeper parts of the tube and forms hazy colonies. It is anærobic, but, unlike *Tr. pallida*, growth may be obtained in the medium without the addition of fresh tissue, although, like *Tr. microdentium*, the growth is poorer when the tissue is absent. In pure culture it measures on the average 0·5 to 0·75 micron in width and 6–24 microns in length. Chains of two, three, or four individuals may be seen. The number of curves varies, but, as a rule, the middle part of the body is merely wavy, while the extremities are more regularly and deeply curved.

The ends of the organism are pointed and to one or both of them is usually attached a fine, long, more or less stiff but elastic projection, which has regular minute curves. The body was thought by Schaudinn to be ribbon-like, but in culture it appears to be cylindrical. The author has not come to any definite conclusion as to the method of division, though the ultimate division of the chains is transverse. No lesions have been produced by its inoculations into monkeys or rabbits.

The organism belongs undoubtedly to the genus *Treponema*, but can readily be distinguished from other members of the genus.

It is supposed by some that *Sp. refringens* and *buccalis* are identical. The author has experienced a greater difficulty in growing *Sp. buccalis* in pure culture, but in impure culture he has been much struck with its resemblance to *Sp. refringens*. The matter, however, requires further study.

C. M. W.

Lieut.-Col. Sir William B. Leishman, F.R.S., on Cell-inclusions in the Blood of a Case of Blackwater Fever.

[Journal of the Royal Army Medical Corps, May 1912, no. 5, pp. 493–504.]

In three blood-films received from a case of Blackwater Fever the author found certain large mononuclear cells (average diameter 25 microns) which he considers to be endothelial cells disrupted from the walls of blood-vessels or lymphatics. Similar cells were described as endothelial cells by Christophers and Bentley in this disease. The nucleus of the cell was generally laterally displaced and often pressed against the side of the cell. The cytoplasm of some of these cells contained granules very unevenly distributed and resembling the neutrophile granules of the ordinary polynuclear leucocyte except that they were smaller. The chief interest is the occurrence of certain cell inclusions in the cytoplasm of these cells. They range in diameter from 1–5 microns, show a tendency to be circular in contour, and have a tendency to stain deeply at the periphery and more faintly at the centre, though the smaller forms are homogeneous. In three instances large forms showed a deeply staining peripheral capsule with a dark central granule. These bodies, which were found only in about a couple of dozen cells, are figured in a plate which shows clearly their characters.

The possible nature of the bodies is discussed under the headings of Artefacts, Products of Nuclear and Protoplasmic change, Phagocyted Material and Micro-organisms. The author sees no possibility of their being artefacts and does not think it likely that they represent products of nuclear or protoplasmic change, since the nuclei of the cells showed no sign of karyolysis or karyorrhexis. However, he admits that this cannot be absolutely excluded. He is perfectly familiar with phagocyted red cells and does not regard this as a possibility, so that he inclines to the view of their being parasitic in nature and related to the Chlamydozoa, a group of organisms found in variola, vaccinia, trachoma, molluscum contagiosum, and bird epithelioma. The author gives a short review of the group, and concludes his article by stating that his main object has been to call attention to these bodies in the hope that further search may demonstrate whether there is any foundation for his suspicion—"it is little more"—that they represent an invasion of the endothelial cells of the viscera and blood- or lymph-vessels by parasites of the nature of Chlamydozoa. C. M. W.

C. França on the Hematozoa of Moles.

[Sur les Hématozaires des Taupes. Archivos do Instituto Bacteriologico Camara Pestana, tome iii. 1912, pp. 271–278.]

The author describes certain hematozoa which he has encountered in the moles of Portugal (*Talpa europæa* and *T. cæca*).

1. Trypanosoma talpæ, Nabarro.

It is pointed out that Laveran and Mesnil state that Gros first saw a trypanosome in the mole in 1845, and it was not rediscovered till Petrie in England found it in six moles in 1905. In 1906 Thomson gave the first detailed description of the trypanosome, and Nabarro named it *Trypanosoma talpæ*. The moles of Portugal harbour the same trypanosome as those of England. It is a very mobile and flexible trypanosome; is characterized by its width, the smallness of its trophonucleus, and the extreme length of its pointed non-flagellate end. The authors have never encountered a large infection. The trypanosomes met with in Portugal appear a little shorter (17 μ) and a little broader (1·9 μ) than those of Thomson in England.

2. ELLEIPSISOMA THOMSONI, n. g., n. sp.

This parasite was observed by Thomson in England in 1906. It is a parasite devoid of pigment and occurs in the red corpuscles of the mole. The percentage of infected moles is much greater in June than at any other time of the year. The parasite is generally elliptical in shape and then measures 4·5–6 $\mu \times$ 3–3·5 μ, but it may have any other form quadrangular or quite irregular. The nucleus of the parasite is usually excentrically situated and possesses no nuclear membrane. The infected red cells are much hypertrophied. Young forms are very rare in the blood, and multiplication forms were only seen in the lung smears. The young forms occurred as elongated vermicules (8 $\mu \times$ 1·5 μ) with a stretched out nucleus, or oval elements (3·5–4 $\mu \times$ 2–3 μ) with a compact nucleus. Multiplication takes place either by simple division into two parts or by a process of schizogony into a variable number of daughter cells. The reproducing forms are extracellular. The protoplasm of the parasite sometimes shows peculiar granules which stain very deeply, but this is not pigment, as the author determined by the examination of fresh specimens.

The relation of the parasite to the Hæmogregarines and the Toxoplasma is discussed. In the former there is a schizogony in cysts, while the latter are parasites of the internal organs and not in red cells; hence the author has decided to create the new genus at the head of this section.

3. ENDOGLOBULAR PSEUDO-HEMATOZOA.

The author has encountered the intracorpuscular bacilla-like rods which were first seen by Graham Smith, who thought they were parasitic in nature. The same rods were found by Thomson, and later Balfour described similar bodies from the jerboa, and the present author in *Microtus incertus* and *Eliomys quercinus*. The red corpuscles may be filled with these structures. Laveran expressed the belief that they were due to anæmic changes in the red cells, but the author cannot trace any connection between their occurrence and any anæmic condition. C. M. W.

SOME REMARKS ON THE SUCCESSFUL INOCULATION OF *LEISHMANIA TROPICA* TO MAN.

BY C. M. WENYON, M.B., B.S., B.Sc.,
PROTOZOOLOGIST TO THE LONDON SCHOOL OF TROPICAL MEDICINE.

DURING the month of August of last year I was conducting experiments in Aleppo, Syria, on Oriental Sore and its transmission. Both there and previously in Bagdad I had allowed flies (*Stegomyia*, *Culex*, *Phlebotomus*) to feed upon sores, and afterwards upon my arm. As no positive result was obtained it was thought that I might possibly be immune to infection. Accordingly some material was obtained from an ulcerating sore by scraping and placed upon my arm, which was scarified with a needle. Some of the material was placed on another spot without scarification and allowed to dry, in order to determine if the leishmania might pass through uninjured skin. It may be stated at once that at the latter spot nothing resulted. On the scarified area, however, there quickly developed a suppurating focus of about 1 cm. in diameter and surrounded by much inflammation. There was discharge of pus, but eventually a scab formed and healing took place beneath this, leaving a slightly pigmented patch in which two small red spots could be detected. These subsequently disappeared.

Having returned to England, six and a half months later I became ill and suffered for about one week from fever up to 103° F., aching in the limbs, and some gastro-intestinal disturbance. I concluded that I was suffering from influenza, but in the course of the week I noticed that a small red papule had appeared at the site of the original inoculation. This increased in size and has grown slowly since then, while two smaller papules have appeared, both within the area covered by the original inoculation. The largest has now been growing for three months and is only about ·25 cm. in diameter. Numerous leishmania have been found in these.

There arise from this experiment several interesting results.

1. *Leishmania tropica*, apparently, cannot pass through the uninjured skin.

2. The long incubation period of six and a half months means that the local development of the parasite is very slow. In a recent paper Patton has shown that the period of incubation after such inoculation may be only sixteen days, and concludes that it varies probably with the number of parasites inoculated. In another case inoculated by me in Bagdad the incubation was two months, which appears to be the more usual period.

3. At the time of appearance of the sore there was constitutional disturbance with fever and other symptoms. Was this the direct result of the development of the leishmannia or did the disturbance of health aid the parasite in its multiplication? Several observers have recorded associated disturbance of health, and Marzinowsky, who inoculated himself, noticed similar symptoms at the time of appearance of his sore. On the other hand, Nicolle records the case of a man who was inoculated with *Leishmania tropica*. Nothing had resulted when seven months later he suffered from Undulant Fever. During the course of the illness, seven months after inoculation, a typical sore developed. It appeared as if the attack of Undulant Fever had lowered the man's resistance.

4. At the time of inoculation there was rather extensive suppuration, which might have been supposed sufficient to destroy the leishmania introduced, which were not, as a matter of fact, very numerous in the material used. The result demonstrates that a suppurating process does not necessarily prevent the development of the *Leishmania tropica* in the skin.

5. The ordinary discharge from an ulcerating sore, containing as it does large numbers of bacteria and other saprophytic organisms in addition to the leishmania, is capable of producing a sore if brought on to any open wound or abrasion of the skin. This must happen occasionally by accident in autoinoculations, and also probably through the agency of house-flies transferring material from a sore to some open wound.

P. C. FLU ON THE ETIOLOGY OF GRANULOMA VENEREUM.

[Die Ätiologie des Granuloma venereum. Archiv für Schiffs- und Tropen-Hygiene, Band xv. Nov. 1911.]

IN this paper the author reviews the history of the disease. He points out that it was first described by Daniels in British Guiana. It is a disease which is venereal in character and attacks both sexes, usually between the years of 18 and 50, and presents itself in the form of red masses of granulation tissue which bleed readily and exude a thin serous fluid of peculiar odour. At the edges the granulations are covered by masses of hypertrophied epithelium raised well above the surrounding skin. There is a tendency to spontaneous healing, but this is generally limited to one part while the disease is extending at another. The disease commences as a small cell infiltration in the superficial layers of the corium, producing an epithelial cone which hypertrophies and extends. Blood-vessels are formed in this tissue, fibroblasts appear, and it is converted into granulation tissue, by the swelling and œdema of which the papillæ of the skin become flattened. The covering epithelium absorbs fluid, and between the epithelial cells which are forced apart there appear polynuclear leucocytes. The epithelium becomes more and more atrophic till finally it disappears, leaving the red bleeding granulations exposed. The infiltrating cells are chiefly plasma cells, and on this account Siebert classes the disease in the Plasmone group, which is a class of infectious tumours related to the rhinoscleroma. They are all characterized by showing beneath the epithelium plasma cells in a layer of varying thickness. Giant cells are not found in the disease.

As regards the cause of the disease various organisms have been described, but it is probable that a variety of infections have been described under this one heading. Thus MacLennan found a spirochæte resembling *Sp. refringens*, Wise spirochætes resembling *Sp. pallida* and *refringens* in association, suggesting some connection with syphilis. Maitland also discovered a spirochæte of the *pallida* type, and described the disease as occurring in the mouth. Cleland found another and thicker spirochæte in the fibrous walls of the blood-vessels and named it *Sp. aboriginalis*. Le Dantec inoculated a marmot with tissue from the disease, and produced tubercles which led him to regard the disease as a variety of tuberculosis. Siebert, in 1907, described an organism from preparations made in both Madras and New Guinea. This appeared sometimes as an elongated coccus, usually arranged as a diplococcus surrounded by a capsule, and at other times as a small rod staining more deeply at the poles. The cocci were at times embedded in a gelatinous mass and occasionally resembled yeast cells. The organism occurred in the plasma cells, which were in consequence much altered. As the parasite was found in large numbers, especially in situations where the disease was still active, and occurred in the disease from different parts of the world, Siebert concluded that it was the cause of the disease. Later, Donovan described a body which he found in cells of the *Stratum malpighii* and in *macrophages*. It stained with Romanowsky stain, showed an oval chromatin mass, and measured 1·5–2 μ in diameter. Sheube considers this to be the same as Siebert's organism. The author of this paper has made a careful investigation of the matter, and after careful cleansing and removal of tissue from the granulations has made preparations both by dry and wet fixation methods. The result is that in the plasma cells, and also to some extent in mononuclear and polynuclear leucocytes, he has found the parasite which is, in his opinion, the cause of the disease. The organism presents itself as a deeply staining granule surrounded by a capsule. In Giemsa stained specimens the granule is of a dark violet tint, while surrounding it is a layer of deep red staining material, outside of which is a much more deeply staining margin. The granule in multiplication elongates and is finally constricted into two. The capsule then becomes constricted and divided in like manner. Within a single cell there may be only two or three such bodies, or there may be fifty or more. Groups of bodies may be bound together in a kind of zooglœa-mass, often applied as a cap to the cell nucleus which becomes displaced. By rupture of the cell the organisms are scattered and are taken up by fresh cells. When extra-cellular they are quite passive and exhibit no spontaneous movements.

The effect of the organism on the cells is to cause a hypertrophy and vacuolation of the protoplasm, a swelling of the nucleus, and the appearance in the cell of structures which resemble those bodies which appear as reaction products in chlamydozoa infections. These bodies stain blue with Giemsa stain and are often surrounded by the organisms. Their origin is peculiar, as they arise, in some cases at least, from the nuclear remains of leucocytes ingested by the voracious plasma cells, which take up these cells as well as the parasitic organisms. A similar ingestion of leucocytes was shown by Prowazek to take place in the case of vaccinia in the rabbit's cornea.

The author believes his organism to be the same as that seen by Siebert, though this observer only saw the diplococcus form, a result probably of his not having used a wet fixation. He believes the organism to belong to the group of the capsuled bacteria, to which group belong also the scleroma bacillus, the bacillus pneumoniæ of Friedländer, the bacillus ærogenes, and others. Characteristic of this group is the form, lack of motility, gram negative reaction, the ease with which culture is obtained on ordinary media, and the tendency to the production of slime masses in culture. He has attempted culture, but so far without conclusive results.

The author concludes :—

1. In Granuloma venerum occur intracellular, rarely extracellular organisms, which are surrounded by a capsule and embedded usually in a zooglœa-mass.

2. The zooglœa-mass has a tendency to surround the host-cell nucleus like a cap, while the cell reacts to infection by the extrusion of nuclear substance.

3. The Granuloma belongs histologically to the Plasmones, and is nearly related to Rhinoscleroma.

In the reaction of the cells and the arrangements of the zooglœa-masses in them there is a striking resemblance to the effect produced by the Chlamydozoa of Trachoma, Molluscum contagiosum, &c., which are supposed to be Protozoa. C. M. W.

I FIND that Dr. C. L. Strangman, working in Port Darwin, has published in the 'Australian Medical Gazette,' Aug. 21, 1911, and in the 'Transactions of the Australian Medical Congress,' 1911, an account of organisms isolated by him from cases of this disease. It appears that on Löffler's medium two distinct organisms can be grown, and, according to him, to produce the lesions of the disease it is necessary for both organisms to infect the tissues. Both grow as a mycelium and give rise to coccus-like bodies, but in one case these are gram positive while in the other they are gram negative. The gram positive bodies develop in cultures through various stages (half-moon bodies, paired triangular bodies, string of large cocci) to minute motile organisms, each provided with a tuft of flagella (medusa-like bodies). After this stage the culture dies out. The gram negative coccus-like bodies become transformed into Cleland's bipolar staining bodies, which develop into organisms resembling those described by Flu from the plasma cells of the lesions. These bodies grow into a coarse mycelium which segments into a fine mycelium. The culture then dies. If, however, both these organisms are cultivated together in symbiosis from the coccus-like stages, they grow much more luxuriantly, with the formation of clear crystalline plaques and sheaves of crystals. Further, the cultures have not the same tendency to die out, and one finds networks of mycelium interlacing the crystals. The cultures can be carried on by subculture of the mature fungi.

The author claims that he has been able to trace within the cells of the lesions all the complicated stages of growth of the two organisms which he has isolated on the culture medium. If this is ultimately proved correct, it would seem that the organism described by Flu represents only a certain stage in the development of the causative agent of this disease. C. M. W.

III.—DEPARTMENT OF HELMINTHOLOGY.

By R. T. LEIPER, D.Sc., M.B.

Some Nematode Worms from Lagos.

By A. CONNAL,
West African Medical Staff.

The six species of worms described below were found in a collection made from various small wild mammals, birds, and reptiles, by Dr. T. R. Beale-Browne in Lagos, during 1910.

The specimens were sent to the Helminthological Department at the London School of Tropical Medicine. Dr. Leiper suggested that I might devote part of my recent leave to their study. Advantage was gladly taken of this opportunity, and I have to acknowledge further the very kindly and painstaking assistance which was received in his laboratory.

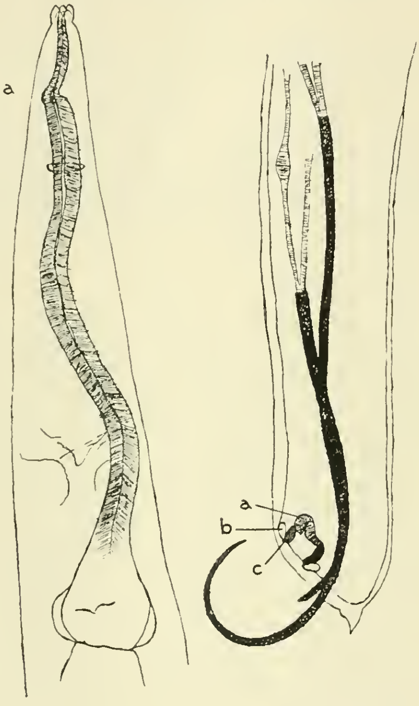
Strongyluris streptœsophageus, nov. sp.

Specimens were collected in large numbers from the stomach and intestine of a male *Agama colonorum*, the common lizard of West Africa.

Male.—Total length 8·6 mm., greatest breadth 0·44 mm. The body tapers gradually to the head end, where the diameter is 0·05 mm. The tapering is less marked towards the posterior extremity, the breadth at the tail end, just before the tiny spike-like termination, being 0·28 mm. The thickest part of the body is at about the level of the œsophageal bulb. The tail end is not coiled or incurved, but is perfectly straight. The cuticle is smooth and devoid of spines or striæ. The mouth is provided with three simple lips arising from a broad base and tapering to a rounded tip. The œsophagus is a slender muscular tube terminating in a well-marked bulb which has three alar folds on its outer aspect. A sinuous course and a sharp kink are two outstanding peculiarities of the œsophagus. The kink is situated 0·23 mm. from the anterior extremity, and the tube takes at least three bends thereafter. The total length of the œsophagus is 1·58 mm., and the breadth increases gradually from 0·04 mm. at the commencement to 0·08 mm. at about its middle, and 0·25 mm. at the widest part of the bulb. The nerve-ring crosses the œsophagus 0·4 mm. from the head, and the excretory bridge is 1·12 mm. from the same point. Slender subcuticular papillæ are seen along the whole length of the body, being more closely set in the anterior third. The anus is situated 0·01 mm. from the tip of the tail. The coiled portion of the testis begins at 1·6 mm. from the posterior end of the worm and continues forward to a point 0·19 mm. from the termination of the œsophagus; it then passes backwards, ending 1·51 mm. from the tail end. The spicules are equal in length, slender, and tapering to a fine point. They are 1·2 mm. long and 0·01 mm. broad. They present a strikingly mosaic pattern. There is a sucker, ventrally placed, 0·76 mm. from the tip of the tail. Its wall in section appears as a comma-shaped chitinous piece, with a small round air-space at the outer rim (fig. B, *b, c*) A muscular band, in the shape of a loop, is attached to the chitinous piece at the inner end.

There are three pairs of pre-anal and six pairs of post-anal genital papillæ. The most anterior pair of the pre-anal set are short and stumpy, the other two pairs being large, long, and finger-like. The anterior two pairs of the post-anal set are long and slender, the next two pairs are short and stumpy, and the posterior two pairs are again long and slender.

Female.—Total length 9·1 mm., greatest breadth 0·5 mm. The general characters of the mouth-parts and œsophagus resemble those of the male. The nerve-ring crosses the œsophagus 0·51 mm. from the head, and the excretory bridge is 1·55 mm. from this point also. The vulva is 3·35 mm. from the tip of the tail. The vagina passes inwards and forwards for a distance of 0·2 mm. and then divides into two uteri which diverge. The topmost coil of the ascending uterus is 0·7 mm. from the commencement of the chyle intestine, and the lowermost coil of the descending uterus is 0·4 mm. from the tip of the tail.



A. B.

Strongyluris streptœsophageus.

A. Anterior extremity.
a. Kink in œsophagus.

B. Posterior extremity.
a. Muscular loop.
b. Air-space.
c. Chitinous piece.

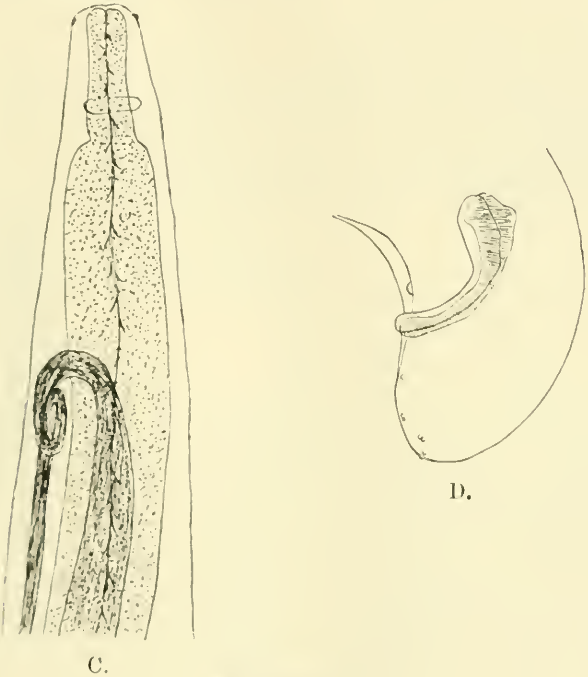
The female is oviparous, the ova measuring 0·06 mm. × 0·03 mm. The eggs are thin-shelled and elongated ovoid. The sucking-disk is absent, as is also the spike-like termination of the tail. The anus is 0·2 mm. from the tip of the tail. The œsophagus is 1·95 mm. long and 0·07 mm. broad at about its middle. The kink occurs at 0·29 mm. from the head.

This species is apparently closely allied to *Strongyluris brevicaudata.*

FILARIA YABA, nov. sp.

Large numbers were found in the thoracic cavity of *Centropus senegalensis*, a very common bird in West Africa.

Male.—Total length 19 mm., greatest breadth 0·6 mm. The diameter of the body is uniform in its greatest extent, a fairly abrupt tapering taking place at the extremities. At the anterior end the breadth is 0·17 mm. and at the posterior end it is 0·2 mm. The tail end is bluntly pointed and is coiled at least 1½ times. The skin is smooth and without bosses or ridges, or striæ. The mouth is simple, and there is a minute papilla on the outer aspect of each of the three lips. The œsophagus is simple and slender, 5·2 mm. long; the first part, 0·36 mm. in length, is only half the thickness of the remainder. This broadening, from 0·11 mm. to 0·22 mm., takes place suddenly, giving the œsophagus a shouldered appearance, like a bottle. The nerve-ring crosses at 0·26 mm. from the head. The chyle intestine, which is of the same thickness as the œsophagus at its junction, widens out very gradually to rather more than twice this breadth, and it ends at the anus, 0·13 mm. from the tip of the tail. The highest coil of the testicular tube is 0·86 mm. from the head, and the termination is 3·4 mm. from the head. There are two spicules which are practically identical in size and shape, their length being 0·24 mm.; they are short, and are broadened and flattened at their origin, narrowing suddenly and bending sharply at the same point, and resembling a boomerang in shape. The genital papillæ are small and difficult to determine. Almost immediately in front of the anus there is one pair, while there are four pairs situated posterior to the anus,



Filaria yaba, ♂.

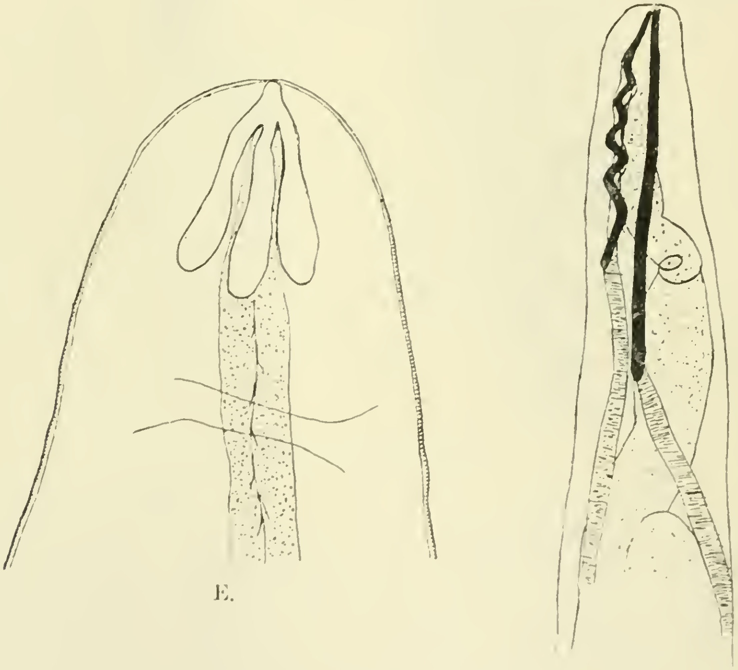
one behind the other. These papillæ are button-like and small, the pre-anal pair being about twice the size of the others.

Female.—Total length 23 to 33 mm., greatest breadth 0·8 mm. Both ends are bluntly pointed, a gradual tapering taking place at the head end, where the diameter is 0·2 mm., and a slighter tapering at the tail end, where the breadth is 0·27 mm. The œsophagus is 6 mm. in length, the first part being 0·12 mm. in breadth and 0·45 mm. long, while the second part is 0·24 mm. thick. The nerve-ring crosses 0·27 mm. from the head end. The vulva is 0·98 mm. from the head. The vagina, broadening from 0·15 mm. to 0·24 mm., passes backwards for a distance of 1·58 mm., where it divides into two uteri which pass backwards side by side. The highest coil of the uterus is 0·67 mm. from the head, and the lowermost coil 0·48 mm. from the tail end. The female is ovo-viviparous, the ova, containing a much-coiled embryo, measuring 0·05 mm. × 0·03 mm. The anus is 0·12 mm. from the tip of the tail.

TRIPLOTRIÆNA FALCONIS, nov. sp.

Numerous specimens from thoracic cavity of small hawk, *Falco* sp.? Wings, head, and back rich brown, chest speckled white and light brown, yellow legs and beak.

Male.—Total length 30·1 mm., greatest breadth 0·46 mm. The anterior extremity tapers to a diameter of 0·1 mm., while at the tail end the breadth is 0·16 mm. The cuticle is very finely striated. The mouth is furnished with three simple fleshy lips, on which no papillæ were seen. Two trilobed



E.

F.

Triplotriæna falconis, ♂.

glandular structures are present, the three lobes uniting to discharge into the mouth-cavity. The œsophagus is slender, varying in girth from 0·06 mm. at its commencement to 0·12 mm. at about its middle, and 0·07 mm. at the constricted part where it opens into the chyle intestine. Its length is 1·78 mm. The nerve-ring crosses it 0·22 mm. from the head. The testicular tube is a broad and prominent structure, its highest coil reaching to 0·47 mm. from the anterior end, and its termination being 6·4 mm. from the head. The spicules are long, narrow, dissimilar and unequal. The larger one is straight, blunt-pointed, 0·9 mm. long, and tapering from 0·024 mm. at its commencement to 0·012 mm. at its tip. The shorter one is corkscrew-like with four distinct twists. It is more pointed than the other and very slightly narrower. No pre-anal or post-anal papillæ were seen. The anus is 0·04 mm. from the tip of the tail.

Female.—Total length 55 mm., greatest breadth 0·6 mm. The mouth-parts, including the two trilobed cephalic glands, and the œsophagus are similar to those in the male. The nerve-ring crosses the œsophagus 0·19 mm. from the head. The vulva is seen 0·37 mm. from the anterior extremity. The vagina passes posteriorly, being 0·4 mm. in length and widening from 0·06 mm. at the vulva to 0·13 mm. at its middle. The two uteri pass backwards side by side, the topmost coil reaching to 0·34 mm. from the head and the lowermost coil 0·36 mm. from the tip of the tail.

The female is ovo-viviparous, the ova measuring 0·062 mm. × 0·046 mm. The anus is 0·36 mm. from the tip of the tail.

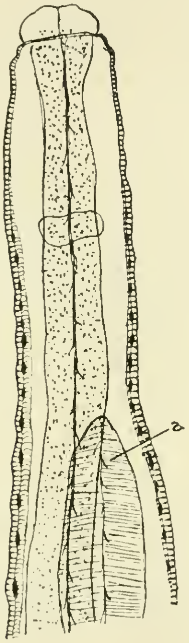
Note.—A similar *Filaria* was found in a collection made from a South African hawk by Mr. G. Norman Hall.

ASCARIS ROSARIUS, nov. sp.

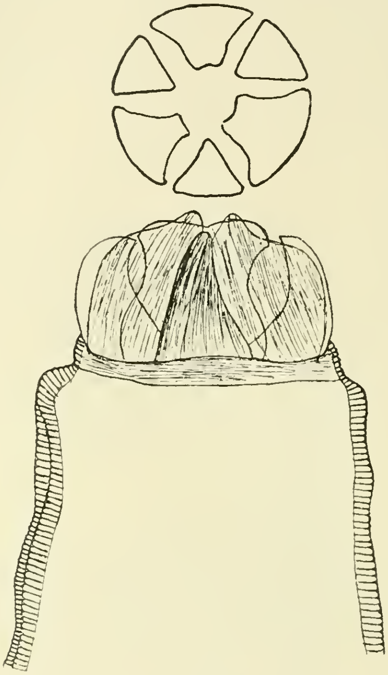
Several specimens, found in the stomach of a small grey heron with greenish plumes on head. *Nycticorax* sp. ?

Male.—Total length 26 mm., greatest breadth 0·6 mm. The body has an elongated spindle shape, but the anterior extremity for a length of 0·7 mm. is narrow and cylindrical, measuring 0·2 mm. in breadth at the head, and maintaining this until it abruptly broadens to 0·3 mm., giving a humped appearance. At the posterior end the body tapers rapidly to a sharp point. The cuticle is strongly striated and numerous regularly arranged refractile dots give a beaded appearance to it, like a rosary, along the whole length. The subcutaneous transverse muscular bundles are especially marked in the posterior half. The tail is coiled ventrally like a pot-hook. The mouth is furnished with six fleshy lips, three of which are large and with their internal and lateral aspects concave. Between each of the large lips is placed a small lip, which is triangular in outline. There is a distinct collar of cuticle round the base of the lips. The œsophagus is 3·9 mm. long, and gradually increases in breadth from 0·05 mm. at its commencement to 0·2 mm. where it joins the chyle intestine. There is a blind prolongation of the œsophagus, 0·7 mm. in length, running alongside the chyle intestine. The chyle intestine itself is continued forwards beyond the junction into a blind pouch 2·46 mm., long, and this pouch terminates at the point where the diameter of the worm increases. The nerve-ring crosses the œsophagus 0·5 mm. from the head end and the excretory pore is 0·01 mm. behind this. The highest coil of the testicular tube is 11·31 mm. from the head. The anus is 0·22 mm. from the tip of the tail. There are three pairs of post-anal papillæ: the first pair are double and button-like, springing from a single base; the second and third pairs are smaller and nipple-like. The spicules are long and have a flanged appearance. They are equal in size and shape. They are thickest along their ventral aspect. They are each 3·28 mm. in length and of fairly uniform breadth, and both taper to a blunt point. There are numerous pre-anal papillæ.

Female.—Total length 30·8 mm., greatest breadth 0·7 mm. The mouth-parts and alimentary system resemble those of the male. The increase in girth occurs 0·97 mm. from the head end. The nerve-ring crosses the œsophagus 0·57 mm. from the anterior extremity and the excretory pore is 0·02 mm. behind this. The vulva is 1·59 mm. from the head. The vagina passes inwards for about 0·17 mm. and then divides into two uteri which run backwards, side by side. The highest coil of the uterus is 6·95 mm. from the head end.



G.
Ascaris rosarius, ♀.



H.
Ascaris rosarius, ♂.

a. Blind prolongation of chyle intestine.

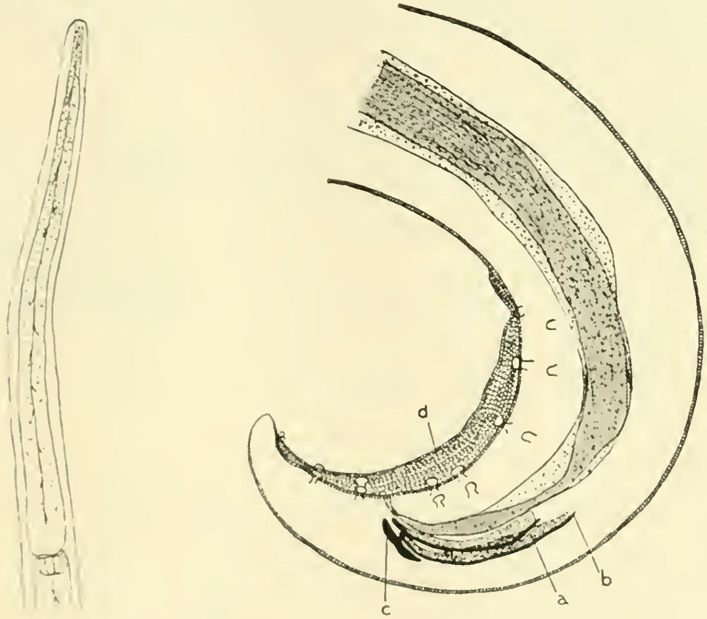
The female is oviparous, the eggs being rounded ovoid, measuring 0·026 mm. × 0·021 mm. The egg-shell is thick.

SPIROPTERA DEFLECTA, nov. sp.

Several specimens from the stomach of the "palm squirrel," *Heliosciurus rufobrachiatus.*

Male.—Total length 19·7 mm., greatest breadth 0·6 mm. Anteriorly the body tapers to a fairly sharp point, the breadth at the head end being 0·08 mm.

The anterior 2·1 mm. is bent laterally at a distinct angle. The tail end also tapers, but more quickly, to a sharp point, the breadth here being 0·04 mm There is a marked curve at the tail like a pot-hook. The cuticle is finely striated, the striations increasing in width and depth from the extremities towards the middle. The mouth is provided with six small lobular lips, an anterior, a posterior, and a lateral pair. The œsophagus is muscular and distinctly bulbous at its junction with the chyle intestine. Its length is 4·15 mm. The anterior portion in front of the nerve-ring is much narrower than the posterior portion. At the head end the œsophagus is 0·06 mm., and at the nerve-ring it is 0·1 mm. The breadth quickly increases to 0·18 mm., then 0·24 mm., and at the bulbous portion the diameter is 0·28 mm. The opening of the œsophagus into the chyle intestine is furnished with three large valvular flaps depending from a collar-like thickening. The chyle intestine continues alongside the testicular tube to the anus, which is situated 0·39 mm. from the posterior extremity. The nerve-ring crosses the œsophagus 0·33 mm. from the head end, and the excretory pore is 0·03 mm. behind this. The testicular tube coils forwards, the highest coil reaching to 4·8 mm. from the head, and the blind termination is at about the middle of the body. The spicules are rather short and broad, and are unequal both in size and shape. The longer of the two is 0·51 mm. in length and 0·058 mm. in breadth, while the shorter is 0·43 mm. long and 0·067 mm. broad. The dorsal part and the tip of each spicule is thickened, while the thinned and broadened ventral portion presents a frescoed appearance. There is a curved



I. K.

Spiroptera deflecta.

a. Short spicule.
b. Long spicule.
c. Accessory piece.
d. Ala.

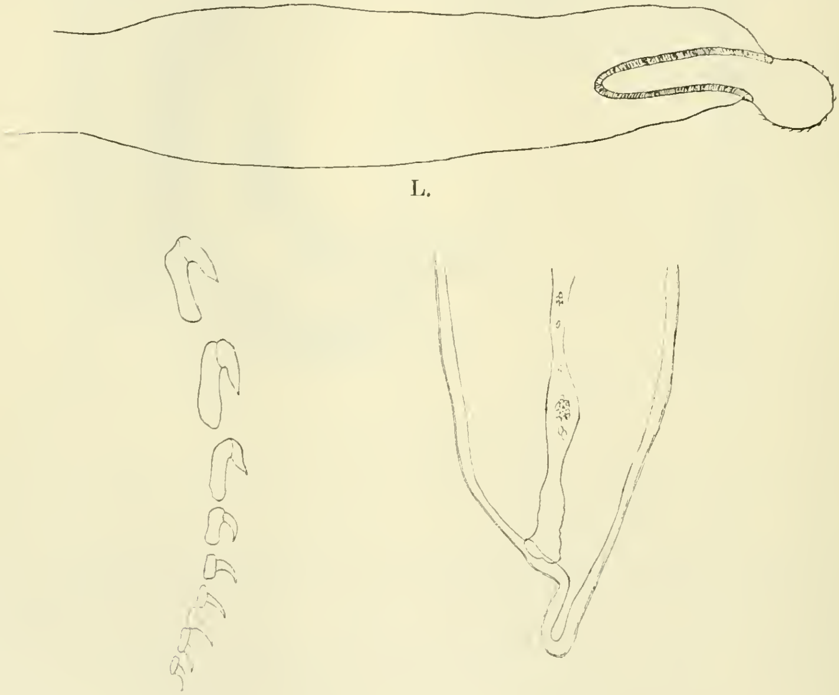
accessory piece, sharply pointed dorsally and blunted ventrally, measuring 0·14 mm. in length and 0·02 mm. in its broadest part. At the tail end, on the ventral aspect, there is a well-marked ala 1·7 mm. long. There are five pairs of pre-anal and three pairs of post-anal papillæ, shaped like mushrooms, each pair arranged behind the other, and all within the alar area.

Female.—Total length 26 mm., greatest breadth 0·9 mm. The anterior portion of the body is bent at an angle like the male, 2·36 mm. from the head. The arrangement of the mouth-parts and digestive tube is similar to that in the male. The œsophagus, which is 5·6 mm. in length, is crossed by the nerve-ring 0·35 mm. from the head, and the excretory pore is 0·43 mm. from the anterior extremity. The vulva is 13·7 mm. from the tail, but owing to the great distention of the body with ova it was impossible to delimit accurately the course of the vagina and uteri. The female is oviparous, the eggs being broadly ovoid and rather flattened on one side, measuring 0·048 mm. × 0·035 mm. The shell is thin. The anus is 0·3 mm. from the tip of the tail.

ECHINORHYNCHUS CENTROPI, Porta, 1910.

Several specimens from intestine of *Centropus senegalensis*.

Male.—Total length 29 mm., greatest breadth 1·2 mm. The worm is cylindrical. The anterior part of the body behind the neck, for a length of 4·9 mm., is swollen out to a thickness of 1·2 mm., but posterior to this the greatest breadth is 0·9 mm. The tail end is rounded and of nearly the same



L.

M. N.

Echinorhynchus centropi.

breadth as the middle of the body. The cuticle is quite smooth. The rostrum is nearly globular, narrowing slightly in front, measuring 0·6 mm. in its longitudinal diameter and 0·5 mm. in its transverse diameter. The hooks are arranged alternately nine and ten in a row longitudinally, and twenty-six in a transverse row. In a row of nine hooks, the five hooks (counting from the neck) are comparatively small, and the blade, which is longer than the handle, arises from the posterior part of it. The anterior four hooks are larger than the others, the blade is smaller than the handle and arises from the anterior part of it. The first hook (counting from the neck) has a handle 0·024 mm. long and a blade 0·036 mm. in length. The terminal hook, at the head end, has a handle 0·09 mm. in length and a blade 0·07 mm. long The neck is short, being 0·38 mm. broad and 0·14 mm. long. The muscular sheath attached to the rostrum is 1·2 mm. in length and 0·4 mm. broad. The testes are 3·2 mm. long and 0·42 mm. in breadth. The cirrus-pouch measures 3·6 mm. in length. The body is strongly muscular, the transverse bundles being especially prominent in the posterior half.

Female.—Total length 48 mm., greatest breadth 1·5 mm. The external characteristics are similar to those in the male, but on a larger scale, except that the posterior end of the body has a finger-like prolongation 0·34 mm. in length and 0·2 mm. broad. The rostrum shows the same number, arrangement, and character of hooks. The uterus measures 2·14 mm. in length and the vagina is 0·4 mm. long.

The female is oviparous, the ova being thick-shelled and of an elongated ovoid shape. The eggs measure 0·05 mm. × 0·029 mm.

IV.—PATHOLOGY.

By H. B. NEWHAM, M.R.C.S., D.P.H., etc.

The Hæmoleucocytic Formula in Carrion's Disease, or Verruga Peruana.

By CARLOS MONGE, M.D. (Lima).

The study of the Hæmoleucocytic variations which occur in this disease was made by me [*] during the years 1908, 1909, 1910, on more than 100 cases: since that time I have continued these investigations and have confirmed the earlier results.

Granted that this disease presents in certain cases a very progressive and marked evolution, and wishing to know the variations which take place at the different periods of the illness. I have carried out systematic examinations of the blood. In fact, nearly every day, in cases of the more severe type terminating rapidly, and in cases of the milder type and of long duration, these examinations have been made every week, or every fortnight, or every month. In this way one can note all the changes which take place in the blood.

In my previous contribution on the subject [†] I have noted two chief clinical forms:—

(*a*) An acute or severe form—nearly always terminating in death, without the appearance of any eruption.

(*b*) An eruptive form, generally mild and of long duration.

One should study these separately, because at the same time the blood characteristics differ considerably.

(*a*) Acute Form.

The most important point noted is the rapidity with which the anæmia comes on from the commencement of the illness. The red cells number some 900,000 per cub. mm. One meets with some cases in which the number falls to 500,000 per cub. mm., the number diminishing up to death, which takes place in a few days. At the same time one notes the presence of microcytes in very great numbers, macrocytes, and the appearance of numerous nucleated red cells: the normoblasts are the most numerous—2000 per cub. mm.: megaloblasts also are found, some 200 per cub. mm. more or less; but in the more severe cases these numbers are considerably increased, and I have recorded one case [‡] in which the number of normoblasts was some 15,300 per cub. mm. and of megaloblasts some 3420 per cub. mm., the case rapidly terminating in death. It is especially in the fulminating cases that one finds a considerable increase in the number of megaloblasts.

One notes also a marked polychromatophilia, not only in the ordinary red cells but also in the nucleated red cells. Poikilocytosis is also marked.

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[*] 'Tesis para el Bachillerato,' 1910, Lima.

[†] 'Journal of the London School of Tropical Medicine,' vol. i. part 2.

[‡] 'La Crónica Médica,' Lima, Perú, 1911.

There are also granular red cells in extraordinary numbers; these were counted on Widal's plan. The nuclei of the red cells present a well-marked variation. In a word, one finds signs of pernicious anæmia. The hæmoglobin value is always increased.

The Leucocytes.

There is always a well-marked leucocytosis. After the first few days of the illness one finds about 20,000 per cub. mm., and these numbers increase with the severity of the disease.

Concerning the relative proportions of leucocytes, there is an increase in the polymorphonuclears, but not very considerable; their number is about 75 per cent. The eosinophiles disappear completely and the mononuclears diminish.

One finds in some cases myelocytes in very small numbers—0·5 per cent.—and also some enormous mononuclear cells like macrophages. I have noted the constant presence of mononuclear cells with protoplasm markedly basophilic, and which resemble the cells one meets with in cases of Sleeping Sickness.

The nuclei of the polymorphonuclears do not show any true lobulation, and, on the contrary, the greater number of the polymorphonuclears show the nuclei simply bilobed in the form of a horseshoe. Perhaps these are young forms, as has been noted in several other diseases (Arnhet—Pappenheim).

This picture of the characters does not change if at this period the eruption makes its appearance; but, on the contrary, one can tell in advance that this eruption will be pale and poorly marked, and it is not uncommon for the eruption to be re-absorbed.

(*b*) ERUPTIVE FORM.

At the commencement of the illness the number of the red cells falls considerably down to, or even below, some 2,000,000 per cub. mm. 1,500,000 is the general rule, but it is admitted that one finds enormous differences in numbers.

Poikilocytosis, polychromatophilia, granular red cells, microcytes, macrocytes, are the chief features which one notes. There are also some normoblasts (less than 1000 per cub. mm.) and megaloblasts (the number of which is extremely small). But there is one very characteristic feature. Serial examinations of the blood show each day a diminution in the signs of anæmia. Thus one sees an increase in the number of red cells, and a diminution in the number of macro- and microcytes, of normoblasts and megaloblasts, and the gradual disappearance of the poikilocytosis and the polychromatophilia, and the same improvement we shall see takes place in the leucocytes when we come to consider the latter.

The Leucocytes.

There is always in the earlier period of the disease a slight leucocytosis. But in order to discover it, it would be necessary to examine the blood during the first few days of the illness, which, however, can rarely be accomplished.

Regarding the different varieties of leucocytes during the first period of the disease, one rarely finds eosinophiles, but one notes that polymorphonuclears are slightly increased. One sees also mononuclear cells with well-marked basophilic protoplasm. But invariably, when systematic examinations of the blood are made, a moment arrives when the leucocytes diminish. The polymorphonuclears fall, the eosinophiles reappear, and a definite mononuclear increase supervenes. It is necessary to draw attention to this point in the most mild cases. There is a kind of true crisis: that is to say, that during an interval of 4 to 5 days the leucocytic formula changes, and this change persists for several days, and even for a month or two.

Hæmoglobin Value.

At the commencement of this form of the disease the hæmoglobin value is increased, but when the mononuclears make their appearance it becomes normal.

In taking a general view of the total changes and variations in the Hæmoleucocytic Formula in these mild cases of the disease, one can sum it up in the following manner:—

(1) *Commencement of the illness.*—Oligocythæmia, microcytes, macrocytes, normoblasts, and megaloblasts (under 1000 per cub. mm.).

Poikilocytosis, polychromatophilia, granular red cells, leucocytosis, slight polymorphonucleosis, mononuclears, with basophilic protoplasm: hæmoglobin value increases.

(2) *Critical period.*—In which the whole blood picture changes completely in a few days or in a gradual manner.

(3) *Decline of the illness.*—Slight oligocythæmia, granular red cells, increase in mononuclears (50 to 60 per cent.). Presence of a slight eosinophilia and the hæmoglobin value normal.

All these points are of the highest importance and are correlated to the clinical manifestations which one notes at the same time, as we shall see subsequently in studying the appended chart. One can thereon note all the points which we have endeavoured to indicate.

It should be noted that the eruption appeared in several subintrant phases. It is on account of this that the critical period of the Hæmoleucocytic Formula lasted from the 22nd of January up to the end of the month. One will note also that a definite increase in the mononuclears was established when the eruption ceased, but a glance at the chart will show all these points better than any amount of explanation.

I should add that in some cases the eruption which appeared in the critical period did not last more than 3 or 4 days. We have seen several examples of such cases, and at the same time I should recall that in the cases of nodular eruption (under the skin) which came out in several crops, this critical period was much prolonged and underwent very notable oscillations. The line which represents this in the diagram is that showing several breaks.

With the establishment of convalescence, only one significant fact has to be noted: an increase in the mononuclears and a slight increase in the eosinophiles is apparent, and this increase in the mononuclears is not even affected by any suppuration.

We have recorded a case in which a septic condition attacked the forearm in a case of Verruga, and, nevertheless, the number of mononuclears was as high as 66 per cent.

From the point of view of the applications of these results to the clinical symptoms, one can draw several conclusions. It is true that in Carrion's disease the eruption is an accident which can happen at any period of the disease.

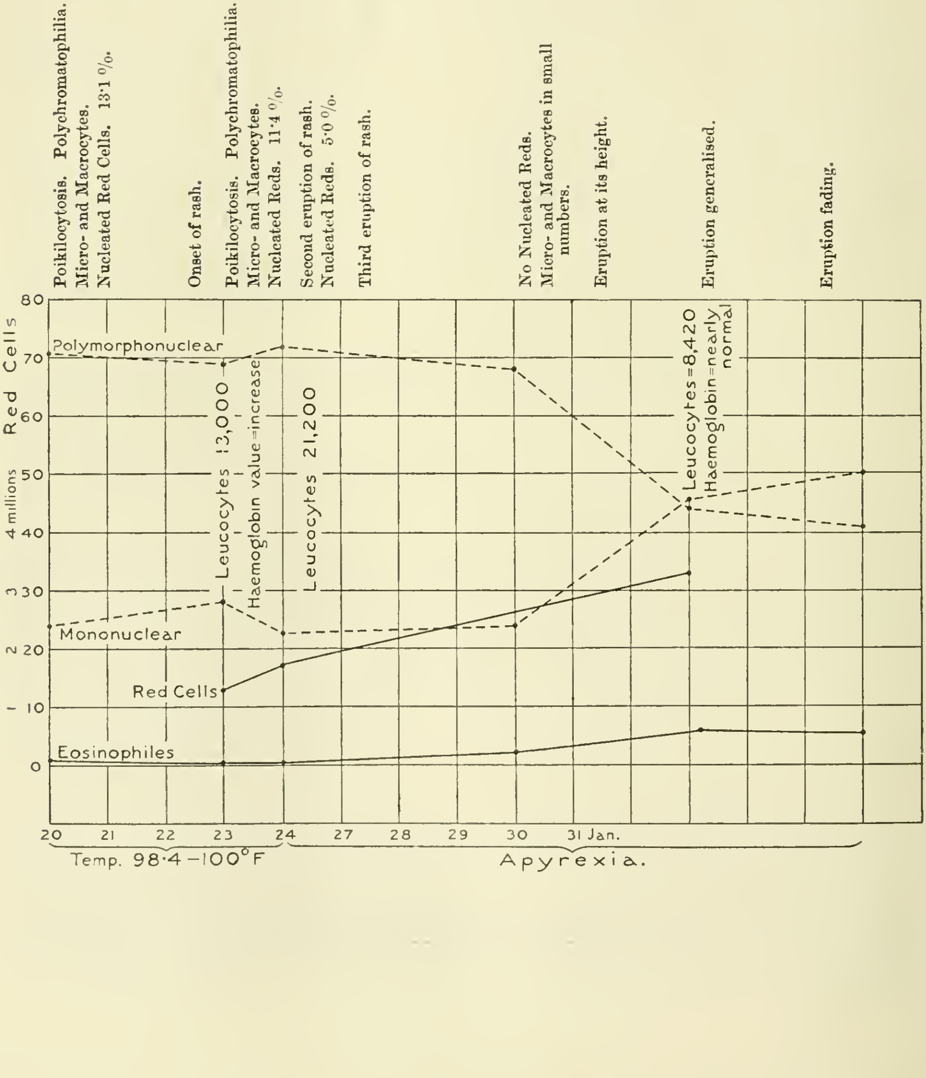
The study of the Hæmoleucocytic Formula and the behaviour of the illness are diagnostic.

The difference between two consecutive examinations of the blood gives the measure of the prognosis.

The increase of the mononuclears and of the red cells marks the amelioration of the illness, so that it is necessary to wait for the eruption.

The appearance of an eruptive rash when the Hæmoleucocyte count shows oligocythæmia, and increase in the polymorphonuclears, together with the variations in the form and coloration of the corpuscles, should not be sufficient to warrant a good prognosis, and it is particularly in those cases that daily examination of the blood gives valuable indications.

In conclusion, it is the study of the Hæmoleucocytic Formula which so controls the progress of the illness that we cannot afford to ignore it if we wish to obtain exact ideas as to the further development of the clinical manifestations in this disease.



A NOTE ON THE PATHOLOGY OF CHYLURIA.

By GEORGE C. LOW, M.A., M.D.

LAST year (July 24th, 1911) I examined the urine of a patient of Mr. Cantlie's who was suffering from chyluria, and found, as Dr. Wise has already described in similar cases in British Guiana, an apparent absence of fat in the milk-like fluid. The attack was evidently passing off, and the sample was not so definitely like milk as in other cases I have seen, the turbidity more resembling the colour of a heavy phosphatic deposit, from which, however, it could be easily differentiated by the usual tests. Moreover, the urine, though standing an hour and a half, did not show any signs of coagulating nor did it separate into the usual three layers. Albumin was present in considerable quantity, and the microscopic examination was as follows:—

No filariæ.
Many small lymphocytes present.
A few red cells.
A few larger cells with fine refractile granules in them.
An epithelial cell here and there.
Calcium oxalate crystals.

The chief cellular constituent of the urine was the small lymphocyte, these cells predominating over all the others and being quite numerous. As to whether the fine refractile granules seen in a few cells represented the remains of fat or not it is difficult to say; but at any rate, even granting this, the amount must have been exceedingly small.

According to Delamere ('General Anatomy of the Lymphatic System'; Leaf's translation of Poirier and Cuneo's 'Treatise of Human Anatomy—The Lymphatics') the difference which is now recognised as existing between the lymph and the mesenteric lymph or chyle is simply that the chyle is loaded with fat derived from the lacteals of the intestine. Lymph, according to where it is taken, may contain traces of fat up to 30 for every 1000 parts, while chyle after a repast contains as much as 65 of fat for every 1000 parts.

The condition of the urine under discussion, then, was a lymphuria rather than a chyluria, the elements present being those of the lymph and not those of the mesenteric lymph or chyle; but what the explanation of this was and what pathological lesions were present to produce such a state of affairs could not, of course, be accurately determined. The block in the lymphatic system was evidently not in the thoracic duct but lower down, where, however, being purely a matter of speculation. The usually accepted idea of the pathology of chyluria is, of course, some obstruction in the thoracic duct with varicosity of lymphatics below the seat of obstruction, the lymphatics in the bladder-wall dilating and rupturing and so pouring the chyle into the bladder.

In January, 1912, a unique opportunity of working out the pathology of what appeared to be a similar case to the above presented itself to me, and the results obtained, results of the very greatest importance, form the basis of this paper. On the 13th of January, 1912, a patient suffering from chyluria and some form of heart-disease was admitted to the Seamen's Hospital, Royal

Albert Docks, under the care of Sir Patrick Manson. The patient was very ill and died while the attack of chyluria was still going on, thus offering one the chance of seeing the exact condition of his thoracic duct, lymphatics, and other viscera. Sir Patrick Manson very kindly allowed me to perform the autopsy and further to describe the case, and my best thanks are due to him for this exceptional chance.

Unfortunately, owing to the serious condition of the patient on admission, it was difficult to obtain full clinical notes about his case, or even to exhaustively examine him, but the following account, taken by the House Physician in charge, brings out most of the requisite points:—

History of Case.

E. S., 24, Ship's Steward.

History.—Patient was born in Brisbane, Australia, and lived there during his youth. After growing up he went to sea as a ship's steward, and in this capacity visited many different parts of the world, New Guinea, South America, &c. His last voyage was from Buenos Aires.

Previous illnesses.—Malaria in New Guinea. Pleurisy 7 years ago on left side. Rheumatic pains in joints, especially in recent years. Never dysentery and denies venereal disease.

Present illness.—Five years ago patient had a fall from a horse and was supposed to have injured his left kidney. After this his urine became white in colour. This whiteness eventually cleared up, but from time to time, especially after any hard exercise, it returned. At one time he was treated for this condition in Australia, after which he came to England, arriving on the 19th of June, 1910, and on the 23rd of the same month he was taken into St. Bartholomew's Hospital as an in-patient, and kept there for 10 months. Feeling relieved after this and the chyluria again having disappeared, he was discharged, and went back to sea, making three trips on a trading steamer. After his last voyage, however, the chyluria returned more severely than before, and signs of heart-disease with vomiting and pains in the epigastrium also having appeared he applied for admission to the Seamen's Hospital at the Albert Docks, and was taken in on the 13th of January, 1912.

State on Admission.—This briefly was as follows: Temperature 96°·8 F. Pulse 92. Respirations 28. Skin cold and clammy; pupils dilated, equal, reactions normal. Considerably emaciated. No enlarged glands or any other signs of filariasis than the chyluria. Marked dyspnœa. Lungs: right apex marked crepitations, some œdema at bases also.

Circulatory system: Heart-sounds very feeble, spacing good, no bruits, no friction. Percussion area enlarged and absolutely dull. Apex beat not palpable.

Alimentary system: Vomiting occasionally, no rigidity of abdomen, marked pain in epigastrium. Liver and spleen not enlarged.

Urinary system: Urine passed with difficulty. Specific gravity 1035. Hæmato chyluria present. On standing about half the amount clotted into a semi-transparent gelatinous mass. No sugar, albumin considerable amount, and blood: no embryo filariæ seen.

Dr. Daniels, who tried to dissolve the clots and milk-like urine with ether, failed to do so, this fact indicating that there was little or no fat present.

Further History.—Signs of pericarditis quickly developed after admission and the patient rapidly sank, death occurring on the 16th of January, 1912. Permission for an autopsy having been obtained, this was begun next day.

Autopsy.—At the suggestion of Sir Patrick Manson the thoracic and abdominal cavities were filled with spirit in order to harden the tissues and also in the hope that the lymph in any dilated lymphatics might be coagulated. In addition I injected the arteries with a mixture of 2 per cent. formalin and spirit and then left the body till the following day.

On the whole, the result of the injection of the arteries and the filling of the cavities with spirit was not successful. The tissues, it is true, were hardened and preserved, but the lymph was not coagulated, and so much general shrinkage took place that the subsequent dissection instead of being rendered easier probably became more difficult.

The autopsy proper was started, then, on the 18th of January, 1912, and was modified in many ways to suit particular requirements, much of the dissection on the tissues removed at that time being carried out on subsequent days.

External appearances.—The body was that of a moderately developed young adult and was considerably emaciated—there were no external wounds or marks such as scars. A hydrocele was present on the right side—the lymphatic glands were not enlarged—there was nothing abnormal about the scrotum—no signs of lymph scrotum—nor were there any varicose groin glands, elephantiasis, or any other external signs of filarial disease.

Head, neck, and spine.—The head and spine showed no abnormalities, the brain and spinal cord being perfectly healthy. As regards the neck, the tongue and mouth were quite normal, there were no enlarged glands in the triangles either anterior or posterior, no signs of inflammation, and the thyroid gland was also apparently normal. The termination of the thoracic duct was as usual.

Thorax.—There were no traces of the thymus. Both lungs were adherent to the pericardium, being bound to it by recent adhesions of fresh lymph. As regards the right lung there were no other adhesions and there was no fluid in the right pleural sac; the left lung, however, presented tough old adhesions at the apex and also laterally. There was no fluid present in the pleural sac. The lungs were then removed and sectioned. Tubercle was present in both: at the apex in the right lung and spreading downwards from that, also at the apex in the left lung, and throughout most of the upper lobe with commencement of cavity formation. The type of the disease was sub-acute with a tendency to acute spread. The lower lobes of both lungs exhibited œdema and some hypostatic congestion. The pericardial area was much increased and was bound to both lungs by fresh adhesions, as mentioned above. The membrane was evidently much thickened. On opening it acute fibrinous pericarditis was present, recent lymph binding the pericardium to the surface of the heart. There was no fluid. At one place on the anterior aspect of the wall of the right ventricle, what appeared to be a small abscess, the size of a pea, was present, and to the right of this a few small granular-like tubercles could be detected. An incision through the wall of the left ventricle showed the myocardium superficially to be infiltrated, and in one or two places to be replaced by a firm caseous-like material. (Sections ultimately showed all these lesions of the pericardium, epicardium, and myocardium to be tubercular—see later.) Further dissection of the heart was not then proceeded with, the specimen being kept intact for museum purposes.

Abdomen.—General examination: No peritonitis, no fluid in abdominal cavity. Bladder full and apparently enlarged. What appeared to be dilated lymphatics on posterior wall of abdomen on left side. Liver and spleen then removed: former weighed 4 lbs. 14 ozs., enlarged, and showed signs of early nutmeg, no tubercle; latter weighed 5 oz., presented the early stages of chronic venous congestion, otherwise nothing peculiar.

Further dissection.—After this superficial examination the remaining viscera beginning at the tongue were removed *en masse*, being stripped off from the posterior wall of the chest and abdomen as carefully as possible, and then dissected in detail.

Thoracic duct.—The thoracic duct was perfectly normal from its termination at the junction of the left subclavian and internal jugular veins right down through the thorax to the receptaculum chyli. Just above the diaphragm there seemed to be a slight dilatation in the calibre, but the walls were quite thin, in no way thickened, and there was no obstruction of any sort. The dilated origin of the duct, the receptaculum, was also normal in every respect. No dilated or enlarged lymphatics could be detected entering it nor did the lacteals in the mesentery appear in any way dilated or abnormal.

Other lymphatics.—On the posterior wall of the left side of the abdomen, running along by the side of the left ureter, which was greatly dilated with a much thickened wall, a dilated lymphatic of considerable size was seen leading down into the pelvis. No increase in size of the lymphatics running towards the lower limbs could be detected, but those of the left spermatic cord were distinctly varicose: those of the right were about ordinary or at the most only slightly dilated; on this side there was a fair-sized hydrocele, however, containing some clear straw-coloured fluid, and in its wall calcified filariæ were ultimately found.

Macroscopically no other dilatation of lymphatics could be detected.

Glands and glandular system.—The glands in the neck were not enlarged. In the thorax there were some slightly enlarged glands at the bronchial roots, but their size was in no way excessive. One long-shaped, apparently enlarged gland, of a somewhat dark black colour, was seen lying beside the thoracic duct in the posterior mediastinum, but otherwise there was nothing to be detected macroscopically. In the abdomen the mesenteric glands were not enlarged nor were the main groups lying in connection with the aorta specially noticeable. On the posterior abdominal wall of the left side, just external to the left common iliac artery, there was a fair-sized gland which cut soft and did not appear fibrosed. A branch of the dilated lymphatic already mentioned seemed to be connected with this.

Both femoral and inguinal groups of glands on either side were carefully dissected out, but none of the constituents of these groups were enlarged. The epitrochlears on both sides were also normal and no other glands of any size were noted elsewhere.

Genito-urinary System:—

Kidneys.—*Left*: The organ was somewhat enlarged; the capsule stripped readily, leaving a smooth somewhat injected surface behind. On splitting the organ through from the outer surface to the pelvis, much reddish tinged milk-like fluid (hæmato-lymph) escaped from its substance. Towards the pelvis this poured out and the pelvis also was full of it. This condition of the kidney indicated that the lymphatics of the organ were in a varicose condition and were discharging lymph, this being one of the sites from which the lymphuria was proceeding. Around and near the pelvis were gaping vessels which were in all probability the dilated lymphatics, this being borne out later in section (see later). No calculi were present either in the kidney or pelvis. *Right*: The right kidney was smaller than the left. On section the same kind of fluid (hæmato-lymph) as was found in the left escaped, but in very much smaller quantities, the lymphatic leakage being much slighter. Calcified filariæ were found in the lymphatics of the pelvis (see later). No calculi were present either in kidney or pelvis.

Ureters.—*Left*: Considerably dilated with a much thickened wall. This condition beginning at and involving the left pelvis extended down uniformly to bladder. The opening into the bladder was quite patent and no cause of any obstruction such as a calculus could be found. *Right*: This appeared quite normal and there was no dilatation of the pelvis. Both ureters were full of hæmato-lymph.

Bladder.—The organ was distended and evidently full. It was opened by a mesial incision, and then hæmato-lymph, similar to that passed during life and to that which was found in both kidneys, poured out. After all the fluid had drained away many large gelatinous clots remained, these having been chiefly responsible for the distension of the viscus. The wall of the organ was much hypertrophied. The mucous membrane was injected. Near the fundus there was an area covered with blood, about the size of half-a-crown, and scattered all over the rest of the general surface were minute little hæmorrhagic spots the size of pin-heads. There was no ulceration of the mucosa and no calculi were present. Next day, after the specimen had been kept in 2 per cent. formalin, most of the blood dissolved away, and then it was seen that the area near the fundus consisted of minute almost villous-like projections extending over a more considerable area than the blood-clot had covered.

Further, where the small pin-head specks of blood had been, now a kind of gelatinous spot remained in their stead. Sections of these areas with their microscopical appearances will be discussed later.

The prostate was considerably enlarged and very hard. In the right lobe a small abscess was present, the purulent collection having a fairly marked capsule around it. Though carefully searched for there were no signs of any adult filariæ in this. Microscopically the pus was seen to contain many eosinophile cells. No filarial embryos were present and there were no tubercle bacilli. No definite coccus either was isolated.

Vesiculæ seminales.—Both the vesiculæ were very much enlarged and stood out as hard prominent masses at the base of the bladder. On section both were seen to be cystic, their entire substance being replaced by well-marked loculated cysts. A thin gelatinous semi-purulent fluid escaped from them when cut. Microscopically this contained lymphocytes, eosinophile cells, and polymorphonuclear cells (pus cells). Many spermatozoa were also present. There were no signs of adult or embryonic filariæ, and no tubercle bacilli were found after staining by Ziehl-Neelsen's method.

The Penis.—As far as could be seen the penis presented no abnormalities, the mucous membrane of the urethra looked healthy. There were no signs of gonorrhœa or of strictures.

The Scrotum.—The tissues of the scrotum were soft and pliable. There was no thickening and there were no signs of lymph scrotum or elephantiasis.

Testicles.—As already mentioned, there was a fair-sized hydrocele present on the right side. Anatomically it presented features similar to any ordinary hydrocele and contained a clear straw-coloured fluid. The testicles themselves looked healthy and were in no way atrophied. There was no special thickening of the epididymis on either side and there were no signs of any abscess formation about any of these parts.

Examination of the hæmato-lymph from the bladder, ureters, and kidneys.—The constituents of the hæmato-lymph taken from the bladder, ureters, and kidneys were similar in nature. Macroscopically the fluid was thick, of a pinkish red to white colour or whitish tinged with pink. On standing some of the fluid separated out into a kind of gelatinous mass of a semi-transparent consistency. The ether test showed that there was very little fat present. Microscopically the following were found:—

No filariæ.
Small lymphocytes not very numerous.
Enormous number of red-blood cells.
Some granular débris.
Some renal cells.
No crystals.

There were no pus cells. The lymphocytes, as already mentioned, were not very numerous, but when cells were met with they were of this nature. Red-blood cells were the predominating feature, exactly the reverse to the case mentioned at the beginning of the paper, where the red cells were scanty and the small lymphocytes very numerous. There was little or no evidence of the presence of fat. The fluid then was lymph heavily mixed with blood; it had not the characteristics of chyle.

General examination for filariæ.—An exhaustive search was made both for adult and embryonic filariæ, the results showing that all active infection had died out, the pathological lesions produced by the filariæ alone remaining. No living adult filariæ were found, but that they had been abundantly present at one time was evidenced by their calcified remains. Dr. Bahr found several in the wall of the hydrocele sac, and I dissected out five from beneath the pelvis of the right kidney. It is almost certain that other similar remains were lying in other situations, but none were actually found. Further, no embryonic filariæ were found anywhere, this again indicating that all the adult forms were dead. Blood from many different situations was carefully searched for these (heart blood, lung blood, blood from carotids, superior vena cava, peripheral blood, &c.), as also lymph from many glands, and the hæmato-lymph from the kidneys, ureters, and bladder. In addition to these the fluid from the hydrocele, the pus from the abscess in the prostate, the fluid from the vesiculæ seminales, and sections of the different organs were also examined.

No embryos were found, as already stated.

Microscopic examination of the tissues.—Many sections were made of the different tissues affected, for microscopical examination. Posterior mediastinal, posterior abdominal, and femoral glands were examined, also the thoracic duct at different levels, the heart, many pieces of both kidneys, the ureters, the bladder and prostate. The small caseous-looking nodule or abscess on the wall of the heart showed microscopically a typical tubercular picture, part of the mass being in a state of caseation, while around this were many giant cells, some of a very large and unusual size. Tubercle bacilli were present in large numbers when the sections were stained by Ziehl-Neelsen's method. Similar lesions were present in the pericardium, and giant cells and fibrosis were also found in the long-shaped dark-coloured posterior mediastinal gland specially mentioned under the heading "Glands and glandular system."—All these lesions, then, were due to the tubercle bacillus and had nothing whatever to do with the filarial infection, so further details as regards them may be omitted. The other glands, posterior abdominal and femoral, showed considerable fibrosis, a great part of their normal glandular tissue being replaced by this; no giant cells were seen in them nor were tubercle bacilli found.

THORACIC DUCT.—All the sections showed the duct to be perfectly normal, no signs of any inflammatory mischief or obstruction being detected.

KIDNEYS.—Near the pelvis of the left kidney there were areas of considerable fibrosis with, in addition, networks of dilated lymphatics, some of the latter being of great size and just visible to the naked eye. (*Vide* macroscopic appearances of kidney.)

Histologically these dilated vessels had all the characteristics of lymphatic vessels. In other sections of the kidney-substance lymph and blood could be seen in the collecting tubules, and in other areas the hæmato-lymph appeared to have forced itself into the connective tissue proper of the organ, disassociating the tubules and parenchymatous tissue generally. No adult filariæ or their remains were seen in any of the numerous sections cut, but some of the calcified adults dissected out from the pelvis of the right kidney were decalcified and sectioned. It then became evident that the worms had been lying in much dilated and thickened lymphatic vessels, and not free in the tissues generally. The characteristic feature of these vessels was their dense thick fibrous walls, masses of fibrous tissue extending out from them into the surrounding tissue and also through their lumen, breaking them up into compartments in which the calcified and broken down remains of the filariæ lay. Judging from the dense thickening the presence of the *F. bancrofti* must act as a very intense irritant, and by producing such extensive fibrosis it is easy to appreciate how permanent obstruction to lymphatics may be set up. The lymphatics infected must have been quite small ones, the little calcified masses only measuring something like 3 to 4 mm. in length by 2 mm. in breadth. The thickest part of the external wall of the lymphatic in one of the sections was ·3 mm., while in other places it varied from ·2 to ·07 mm. Calcification had proceeded to an extreme degree, little or no traces of the soft parts of the worms remaining. Death most likely must therefore have taken place a considerable time before the demise of their host, the patient.

Ureter.—The right ureter presented nothing abnormal. The left, which, as has already been seen, was greatly hypertrophied and dilated, showed microscopically a general hypertrophy of its coats, the sub-mucous coat being much increased in size and showing dilated spaces which were in all probability dilated lymphatics. The epithelium lining seemed fairly normal.

Bladder.—Sections taken through the area which showed the villous-like projections exhibited a somewhat remarkable and peculiar appearance. The mucous membrane was hypertrophied and raised up in a series of folds or definite villous projections covered with the ordinary bladder epithelium. Underneath the epithelium was a small-celled infiltration which passed down into the sub-mucous layer, which was very considerably increased and contained numerous vessels—arteries, veins, and lymphatics. The latter were very numerous and consisted of a network of thin-walled spaces very similar to those seen in the kidney. Most marked near the commencement of the muscular layer, others nevertheless were definitely in the sub-mucous layer, and certainly from some, apparently from others, numerous newly-formed capillaries ran up through the mucous layer and opened on the surface of the bladder at the tips or extremities of the villous-like projections. The appearance of these capillaries was very similar to those seen in ordinary granulation tissue; and the question at once suggested itself, "were these arterial or lymphatic capillaries?"

Poirier and Cuneo state that the trend of modern opinion is to deny the existence of absorbent vessels (lymphatics) in the vesical mucous membrane, and to maintain that the only network of origin which exists in the bladder is an intramuscular one. That this is so in normal bladders is quite likely, but, then, in this individual instance one was dealing with an obstruction to the flow of lymph from the bladder and a consequent dilatation of the lymphatics—as clearly seen in the sections; and there is no inherent impossibility that in such a case new lymphatic capillaries might not be formed and even burst forth through the mucous membrane and discharge their lymph into the cavity of the bladder.

Something of this sort must take place in cases where the escape of chyle or lymph is from the bladder alone.

In favour of the capillaries being arterial, however, was the fact that when the bladder was first opened the area from which the sections under discussion were taken was covered with freshly effused blood, indicating that blood was certainly being poured out or oozing out as well as lymph. The newly-formed vessels did not show red corpuscles in them, however. The muscular coat of the bladder was also much hypertrophied. Sections from the areas where the pin-head specks of blood were seen when the bladder was opened showed a somewhat similar appearance to that described above. Elsewhere the bladder was more or less normal. There was certainly no suggestion of anything tubercular about it.

Prostate.—Sections did not help in revealing the cause of the abscess in this organ. The microscopic appearances of the pus, &c. have already been described.

Discussion of Case.

An analysis of the different points brought out in these two cases of so-called chyluria at once demonstrates the fact that it is altogether erroneous to call all cases of milky urine chyluria. These two cases were in reality cases of lymphuria, the second one in addition showing much blood, *i. e.* a condition of haemato-lymphuria. This term of lymphuria will have to be used therefore for similar cases in contradistinction to chyluria in the future. In all probability such cases will be found much more frequent than is generally supposed, Dr. Wise's work in British Guiana, already mentioned, bearing this out.

The escape of lymph in such cases may take place from any part of the urinary tract, from either or both kidneys, from the ureters or the bladder.

In the second of the two cases described above there was a leakage of lymph from the left kidney, probably the left ureter, the bladder, and in a much slighter degree from the right kidney, this wide distribution indicating an extensive disturbance or blockage of the lymphatic stream passing up from these organs to the thoracic duct.

Where did this take place? To appreciate this a study of the lymphatics of the kidneys is necessary. These are now usually described as arising from two networks, one superficial and the other deep. The deep network gives origin to large collecting trunks, varying in number from four to seven, which leave the kidney at the hilum and run round the renal artery and vein. There would seem to be some dispute as to what glands these trunks end in, but Poirier and Cuneo sum up the matter by saying that "the lymphatics of the kidneys end principally in the juxta-aortic glands of the corresponding side, and accessorily in the præ-aortic glands. After passing through these series of glands the efferents of the right side pass through the right pillar of the diaphragm through the same orifice as the great splanchnic nerve and terminate in the thoracic duct. On the left side the efferents pass through the left pillar of the diaphragm and also join the thoracic duct."

An obstruction (stenosis) of these two terminal trunks, or an extensive fibrosis and blocking of the juxta-aortic glands adjacent to the kidneys, could easily, one would think, block the 4 to 7 efferents proceeding from these organs, and then the lymph would stagnate, the lymphatic trunks gradually dilating and becoming varicose, some eventually rupturing and so pouring their lymph or lymph and blood into the tubules of the kidney or direct into the pelvis, and so *viâ* the ureters to the bladder. In planning out such a scheme anastomoses must of course be considered, and that such do take place, or perhaps rather that the complete blocking of these channels is an unusual one, is borne out by the fact of the comparative rarity of chyluria and lymphuria.

The implication of the vesical lymphatics is also easy to understand. The efferents from the bladder pass into the external iliac glands, the hypogastric glands, and the glands of the bifurcation of the aorta. In turn efferents from these glands pass upwards into the juxta-aortic chains, and so obstruction of the latter might quite easily affect some of the collecting radicles in different parts of the bladder. Again, of course, such a combination of circumstances must be very rare.

By far the most common site of filarial obstruction, which so often ends in elephantiasis, is the lower limbs. Such being the case, blockage of the lymphatics supplying these areas must be correspondingly easy, or the lymphatic glands and the lymphatics of the parts must be a site of special selection for the parent worms. The former supposition is more likely the correct one, however. Without going into the subject in minute detail, a study of the lymphatics of the lower limbs and pelvis shows that the efferent vessels of the superficial inguinal glands end either in the deep inguinal or in the external iliac glands. The former are less numerous, the latter are more so and also much more important. Varying from 8 to 12 in number, they pass through the crural ring, and the majority end in two glands—viz., the external and internal retro-crural glands of the external, and internal chains of the external iliac glands. Again, the efferents from the deep inguinal glands eventually also end, or at least almost all end, in the internal retro-crural gland, so that an obstruction and inflammation of these two glands alone by adult filariæ is practically sufficient to block the whole of the lymphatic circulation of the lower limb. That such a thing apparently does happen is evidenced by the frequency of elephantiasis of the lower limbs in all countries where filariasis is common.

Some may say that even though these glands were blocked, anastamoses would develop and easily carry off the lymph. In certain cases this probably does occur; but if it always did so and was common, then elephantiasis should be correspondingly rare, which it is not. Most anatomists hold that the superficial and deep lymphatics are clearly independent of each other, *i. e.* until they join or come together in the crural ring. Superficial anastamoses over the front of the abdomen, penis, scrotum, &c., of the lymphatics from both sides, take place, but the calibre of these capillaries would not be sufficient to deal with the whole of the lymph from such a large area as the leg.

Many points are still obscure about the pathology of the lesions produced by *F. bancrofti*, but a study of the external iliac glands and their anatomical relations will convince most people that herein lies the frequency of filarial lesions of the lower limbs.

Gravity, owing to the dependent position, must also, of course, play a very important part in increasing the œdema when once this has started.

In real chyluria—that is, where the mesenteric lymph or chyle, the product of the digested food from the intestines (which is distinguished by its abundance of fat), appears in the urine—the obstruction must be in the thoracic duct somewhere above the receptaculum. As already hinted, these cases are probably much rarer than lymphurias.

As regards the presence of tuberculosis in the case described above, this would seem to be only a pure coincidence. Dr. Bahr has recently drawn attention to the presence of giant cells in glands infected with filariæ in Fiji, and thinks that the chronic irritation produced by the parasite may produce this condition. This is possible, but in this instance the giant cells were due to tubercle and had evidently nothing to do with the filariæ. It is just as well to bear this in mind, then, in dealing with such cases.

The condition of the vesiculæ seminales previously alluded to is also a very interesting one. So far as I am aware, there is no record in the literature of these organs being affected by the *Filaria bancrofti*, and the same may be said of the prostate. Still, considering the frequency with which the epididymis is chosen as a site by the parasites, there is no inherent improbability in these other two parts of the genital system being also selected. The probability is that in the past they have not been systematically examined for the presence of filarial lesions, and it certainly will repay one to do this in the future.

Opportunities for complete examinations of filarial cases such as the above are not common, and therefore I have described this one in considerable detail, especially so as the pathological lesions found, in addition to materially changing our conceptions of the pathology of chyluria, give us a new term, "filarial lymphuria," and add some other points of interest to our knowledge of the morbid anatomy caused by the *Filaria bancrofti*.

LUNG LESIONS IN LEPROSY.

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THE pathological anatomy of leprosy is now fairly well known. There are, however, some doubts and differences as regards the presence of lesions in some of the internal organs, more especially with regard to the lungs and intestines. While recently engaged in investigating the value of nastin as a treatment of leprosy, it became necessary to perform some post-mortem examinations, and included in the series were two instances in which the lungs were involved and one in which lesions were found in the intestine.

Hillis [*] writes :—"All the authorities agree in stating that the lungs are not affected in leprosy, but that tubercle may sometimes be met with in the pleura."

Scheube [†] writes as follows :—"The occurrence of lepromata in the lung and intestine is disputed by Hansen and Looft, who base their statement on the fact that they have never observed a leprous bronchial or mesenteric gland. On the other hand, Bonome, Doutrelepont, Wolters, and Arning are of opinion that pulmonary leprosy exists, and the case observed by Doutrelepont and Wolters, in which the sputum during life contained numerous bacilli lepræ, should place the point beyond dispute. The lungs in this case appeared to be microscopically healthy, with the exception of small lobular areas in the right upper and lower lobes. These microscopically proved to be peribronchitic infiltrations of the alveolar septa, with numerous cells and globi containing bacilli ; the bronchial glands in this case were typically diseased."

As to the intestine, Doutrelepont [‡] and Wolters [§] in their case found spots under the muscularis mucosa, and Schäffer in one case discovered, close in front of Bauhin's valve, a few somewhat raised whitish-yellow areas with depressed centres which were found to consist of leprous tissue. In this case there was also leproid disease of the mesenteric glands.

G. A. Hansen [‖] in 1903 states that it is not only the skin, the mucous membrane of the nose, mouth, throat, and larynx that are attacked by leprosy, but practically any organ can be the site of leprous disease. The lungs are not often affected : on several occasions they have, however, been found leprotic.

In the cases of lung leprosy which he had seen there was nothing microscopically characteristic to note : some thickening of tissues without any characteristic appearance, at the most a chronic pneumonic condition. Microscopically were found masses of bacilli and globi without special reaction in the surrounding tissues, the alveoli partly filled with cells, many however empty. He never found the bronchial glands affected. He has never seen the intestine affected, though, as he explains, this may be for want of searching.

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[*] Hillis, J. D. 'Leprosy in British Guiana,' 1881, p. 67.

[†] Scheube, B. 'The Diseases of Warm Climates,' 1903, p. 261. Edited by J. Cantlie.

[‡] Doutrelepont. Verh. d. Deutsch. dermat. Gesellsch.

[§] Wolters. Centralbl. f. Bakt., Bd. xiii.

[‖] Hansen, G. A. Handb. d. pathog. Mikroorg., Bd. ii. Kolle u. Wassermann, 1903. Art. "Lepra," p. 187.

Sticker * states that numerous lepra bacilli may be scattered throughout the lungs without there being any microscopic changes.

The Phthisis leprosa pulmonum, incorrectly denied by some or considered to be a complication by tuberculosis, corresponds closely to the anatomical description of the latter disease. In certain cases there is a chronic peribronchitis with accompanying cirrhotic condition of the lungs, in others a bronchiectatic form, and again a caseous pneumonia leading to destruction of the lung-tissue similar to that caused by the tubercle bacillus ; moreover, the presence of phthisis leprosa and phthisis tuberculosa are known in the same patient.

The bronchial glands in leprous affections of the lungs are involved in all cases.

In the intestines Sticker considers that leprous lesions are rare.

Babes †, writing in 1906, sums up the condition of the lungs in leprosy as follows :—

1. The lungs can be quite normal and without any lepra bacilli.
2. The lungs can appear macroscopically and microscopically natural and yet contain lepra bacilli.
3. The lungs may have the appearance of a hypostatic pneumonia or a broncho-pneumonia containing pneumonococci and streptococci, lepra bacilli being absent.
4. The lungs can be the site of tuberculous disease with caseous pneumonia, peribronchial thickening and cavities, tubercle bacilli being present, lepra bacilli being absent.
5. In many of the preceding class neither tubercle, lepra bacilli, nor other micro-organisms were found.
6. In chronic interstitial pneumonia are found peribronchial masses of leprotic nature.
7. The lungs show large areas of desquamative or fibrinous pneumonia of a leprotic nature, with innumerable lepra bacilli. These areas may go on to cavity formation.
8. There are also types of bronchitis and gangrenous bronchiectasis of a leprotic nature surrounded by necrotic areas or by interstitial or desquamative inflammation.
9. There exist cases difficult to doubt in which tuberculous lesions are combined with leprotic lesions.

Babes further goes on to state that the leprotic lesions of the lungs appear very much like those in tuberculosis, except usually in leprosy the anatomical changes are much slower and much more solid ; the tissue is more rarely caseous and there is less destruction and elimination. With reference to the intestine, Babes found ulcers in three cases of mixed leprosy. On the peritoneum behind these ulcers in two cases were found two small sclerotic nodules ; microscopically neither tubercle nor leprosy bacilli were found in these nodules or ulcers.

In the course of pathological investigations of the effect of nastin therapy on leprotic lesions in the human being, a condition of the lung was found

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* Sticker, G. Handb. d. Tropenkr., Bd. ii. 1905. C. Mense. Art. Aussatz o. "Lepra," p. 189.

† Babes, V. Handb. d. pathog. Mikroorg., Ergänz.-Bd. Erst Hft. Kolle u. Wassermann, 1906. Art. "Lepra," p. 174.

which, though resembling tuberculosis, did not exactly correspond to its classical lesions. When the authorities on this subject were consulted, the above discordant views were revealed. During the investigation I therefore turned my attention more particularly to the condition of the lungs, and made careful microscopical examinations whether naked-eye changes were apparent or not. Whenever acid-fast bacilli were found in the lung, portions of the tissue were pocketed in the thigh of a guinea-pig in order to exclude any possibility of the lesion being caused by the *Bacillus tuberculosis.*

In all eleven cases were examined, all of the male sex; in only two, however, were any naked-eye changes discovered. These are described below. Though microscopic specimens of the other nine cases were carefully examined, no acid-fast bacilli were discovered. The number of cases is of course far too small to give any indication as to the frequency of lung lesions in persons afflicted with leprosy. It is of course quite possible that the condition is extremely rare, and that chance led me to two cases in quick succession.

The first case is that of a male aged 17, of black race, born in British Guiana. He had suffered from leprosy for about nine years. The type of leprosy was that of a well-marked nodular infiltration. Numerous nodules were present on the cheeks. The ears had many nodules along the edges, and the lower part was heavy, large, and pendulous. The chin and neck were thickly infiltrated. Many large discoloured patches were present over the back, some showing a papular condition.

Both epitrochlear glands were large and easily felt; the skin over the elbows was thick and harsh, forming a callosity. The ulnar nerves were both stringy; no definite bulbous enlargement was felt. The whole of the forearms, back of the hands, and fingers were involved in a general diffuse thickening, which was hard and rough and did not yield to pressure. It consisted of one mass of leprotic infiltration. The first finger of the left hand and the fourth and fifth fingers of the right hand showed large superficial ulceration extending almost to the base of the fingers. The femoral glands were hard and enlarged, sometimes tender. The legs below the knees were hard, rough, and enlarged. There was ulceration of both great toes, and on the right foot a perforating ulcer over the head of the third metatarsal bone.

There was complete anaesthesia of both hands and feet, with inaccurate perception in the forearms and legs. Complete anæsthesia was present over the maculopapular patches of the back.

In the course of treatment with nastin severe and extensive ulceration broke out all over the body where leprotic lesions existed, leading to severe asthenia and finally death.

There were no leprotic lesions of the mouth or throat. No lepra bacilli were found in the saliva, though the examination was carried out on three separate occasions.

The body was extremely emaciated; superficial ulceration was widespread on all four limbs and on the face and ears. The discharge from the ulceration had dried to form a scabby covering.

The brain was not examined. The organs of the mouth, naso-pharynx, and neck were natural. The right and left lungs weighed 10 ounces; each had petechial hæmorrhages in the substance. Slight anthracosis, but no gross pathological lesion. The heart weighed 5 ounces; the external fat had changed to a gelatinous material, the muscle was pale and flabby, the cavities dilated. The spleen weighed 6 ounces, was slightly blackened with malarial pigment (not recent), and showed some diffuse irregular fibrosis.

The kidneys weighed 4 ounces each. The cortex was profusely streaked with red. The medulla was pale, while the capsules stripped readily. Microscopical sections showed great congestion and an acute tubular nephritis.

The abdominal cavity and intestines were natural. The liver weighed 48 ounces, was in an early nutmeg condition, also fatty in consistence.

Microscopical sections of the petechial hæmorrhagic spots showed a diffuse small round-celled infiltration with occasionally a giant cell. No fibrosis, but a slight catarrhal cellular exfoliation in the surrounding alveoli. The blood-vessels and capillaries were greatly congested. No increase of leucocytes or inflammatory exudation. There were numerous leprosy bacilli present, apparently all intracellular.

A piece of this lung including a petechial spot was pocketed in the thigh of a guinea-pig. After six weeks there was no sign of tuberculosis.

Sections of other organs showed lepra bacilli present in the liver and spleen.

The second case was that of a man of black race, aged 24 years, born in British Guiana. He had suffered from leprosy for thirteen years. This was a case of nodular leprosy. The hair on the eyebrow was absent. There was general infiltration of the cheeks, forehead, and chin, but no definite nodules. Both ears were slightly thickened. The forearms showed many nodules irregularly scattered : the hands were thickened all over with raised plaques of infiltration on the dorsal aspect. The fingers showed small areas of ulceration.

The epitrochlear glands on each arm were enlarged and hard. The ulnar nerve was not felt. The legs below the knees were thickened and harsh. The toes were ulcerated.

There was slight feeling with inaccurate perception in the forearms, hands, feet, and legs. In some parts total anæsthesia was present. Subsequently much of the infiltration ulcerated and considerable wasting took place. No lepra bacilli were found in the saliva ; in the fæces, however, many lepra bacilli were present in large clumps readily found. The ulceration continued to spread over all the leprotic areas, great wasting set in, mild insanity appeared for a few weeks, and finally with great emaciation death closed the scene. On several occasions the fæces were examined, and each time lepra bacilli were readily found.

The post-mortem examination showed that the body was scaly all over, thin and emaciated. All leprotic infiltrations were ulcerated and the exudation had dried, forming a scabby covering. The hands and feet were extensively and deeply ulcerated. The organs of the mouth, naso-pharynx, and neck were natural. The lungs both showed small areas of bronchial consolidation. Many pinhead nodules of a whitish, solid, semitransparent character were scattered throughout the substance of both lungs. No caseation was observed. The bronchial glands were swollen and hard, but not confluent. No nodules were found on the pleuræ. The heart was fibrotic ; no valvular lesions. The spleen weighed 12 ounces, was red and firm. Numerous small nodules were present throughout the substance, some of which were caseous. The kidneys were deeply congested and had one small white nodule in each cortex. The whole of the peritoneum was covered with small nodules, which also were present in the walls of the intestines. No ulceration of the mucous membrane of the intestine was found.

The liver weighed 45 ounces and contained many small and caseous nodules. The femoral glands, with the lumbar and retrothoracic glands, were all enlarged and contained much white hard new growth.

Parts of caseous nodules from the liver and spleen were put in the thighs of guinea-pigs. A portion of the lung containing two of the small nodules was also put in a guinea-pig's thigh. In no instance was any sign of tuberculosis found when killed after six weeks. Microscopical section of the liver showed congestion and œdema. Early cirrhosis in the portal canals was present, though Glisson's capsule was not thickened. The necrotic areas were surrounded by a small round-celled infiltration. Lepra bacilli were found both in the necrotic masses and in the round-celled infiltration.

There was no marked fibrosis in the spleen. The larger areas of necrosis were surrounded by smaller subsidiary areas, which were circled by a small round-celled band in which were frequently noticed giant cells. Lepra bacilli were found in large quantities in the necrotic masses.

The kidney was affected by cloudy swelling. Some glomeruli were obliterated. There were no interstitial changes. No lepra bacilli were found. The pancreas, on microscopic section, appeared natural; no lepra bacilli were found.

The skin near the ulcerated surfaces showed great exfoliation of the epidermis, which was in consequence thin; there was a lack of pigment in the lower layers, and the papillæ were well marked. Some dark pigment was present in the dermis. The globi cells were numerous and packed with brown granules. Many masses of the brown granules were loose in the tissues; this might possibly be due to manipulation in the preparation. Enormous masses of lepra bacilli were present all over the dermis and showed extensive advanced bacteriolysis.

The peritoneal nodules were composed of thickened areas of round-celled infiltration with necrotic centre. Few lepra bacilli were found. Similar nodules in the intestinal walls (muscular layer) were packed with the lepra bacilli.

The lung nodules were areas of necrosis surrounded with small round-celled infiltration in which was occasionally found a giant cell. In these necrotic areas lepra bacilli were readily found. Many smaller petechial areas containing lepra bacilli were also present, but invisible to the naked eye. The femoral, lumbar, and bronchial glands were all of similar construction. Extensive fibrosis was present, with scattered necrotic unstained areas. Many very large giant cells, with nuclei as a rule in the centre of the cell, were found sometimes three or four in the field at once. These giant cells are not definitely related to the necrotic areas. Many eosinophiles were also noted. Lepra bacilli were found in all the necrotic patches, but not in the giant cells.

Both these cases were at the time of death in an advanced stage of the disease, and it is possible that the lesions in the lungs, which were certainly of a comparatively recent character, partook more of a final general dissemination than of a natural sequence of leprosy. From general clinical observations I am inclined to think that such cases as these do not occur until the disease is greatly advanced. The second case has many close resemblances to the general miliary tuberculosis in this country. Miliary tuberculosis in British Guiana often presents itself with miliary tubercle in the lungs and peritoneum and caseous nodules in the liver and spleen.

Tuberculosis is, however, completely negatived by the experiments on guinea-pigs. Moreover, though the microscopic lesions have a superficial resemblance to those of tuberculosis, the arrangement of the cells, the central nucleated giant cells, the clumps of numerous bacilli instead of scattered single ones, all point definitely away from infection with the *Bacillus tuberculosis.*

It is important to recognise, however, that the miliary type of leprosy is not an acute early manifestation of the disease, but a late super-charging of the leprosy bacilli.

THE RELATIONSHIP OF THE ACID-FAST BACILLI.—F. Wills has examined the relationship of the five following acid-fast organisms:—(1) Tubercle bacillus; (2) Leprosy bacillus; (3) Acid-fast bacillus from urine (not tubercle, ? smegma); (4) Timothy-grass bacillus; (5) Tubercle-like bacillus from the blind-worm. The two methods employed were complement-fixation tests, qualitative and quantitative, and determinations of the opsonic index.

He notes that both tuberculous and syphilitic antigens may give complement-fixation with leprous serum, but holds that these are non-specific reactions and that leprous serum has a great tendency to give complement-fixation with any reactive antigen, and considers that leprous serum gives specific complement-fixation, particularly if the method is made quantitative.

His conclusions are:—

1. There are definite substances common to all acid-fast bacilli.
2. These substances are able to produce specific anti-bodies.
3. They belong to the fatty group (fatty acids), lipoids and neutral fats.
4. An organism that has been in contact with the virus of tuberculosis not only gives a reaction against substances of the tubercle bacillus, but even against those of other pathogenic and non-pathogenic acid-fast bacilli with but quantitative differences.
5. In the same manner an organism under the influence of leper virus not only reacts against the substances of leper bacilli, but also against substances of other acid-fast bacilli, chiefly those of tuberculosis.
6. The relationship of all acid-fast bacilli is established by the fact that their characteristic chemical bodies can be extracted from the bacilli, and these bodies have to be regarded as the property which these organisms possess in common.
7. With regard to leprosy, anti-bodies are produced more against the fatty acids and lipoids than against the neutral fat. This fact explains the therapeutic efficiency of nastin as causing the formation of fatty anti-bodies. With regard to tuberculosis, the formation of anti-bodies seems to be somewhat different from what we have seen with leprosy (Much and Leschke!).
8. The existence of the anti-bodies against neutral fatty bodies that have been discovered by Much is very important. They even exist in a much larger amount than Much himself at first thought.
9. All the non-pathogenic acid-fast bacilli show nearly normal opsonic index. With pathogenic acid-fast bacilli the opsonic index is altered.

There exists a remarkable reciprocity in the behaviour of leper and tubercular sera against the bacilli of leprosy and tuberculosis.

10. All these reactions against acid-fast bacilli are to be regarded as entirely specific.

Centr. f. Bakteriol. 1te Abt. (Originale) Bd. lxi. Heft 1/2, S. 37.
R. T. H.

ETIOLOGY OF BERI-BERI.—Chamberlain and Vedder have continued their studies on beri-beri. They believe that polyneuritis gallinarum and beri-beri are essentially the same disease, both being produced by a deficiency of some as yet unknown substance in the food, and in a previous communication they showed that the neuritis of fowls could be prevented by giving an extract of rice-polishings. The extract of rice-polishings contained 1·34 per cent. solid matter, of which 0·03 per cent. was ash. The latter is probably negligible, as it consists entirely of inorganic constituents, chiefly of lime, magnesium, and potassium carbonate. These salts were tried and were not found to have any influence on the polyneuritis. Nitrogenous matter comprised 0·04 per cent. of the total solids. Of this only 0·02 per cent. was present in the diffusate, which was shown to contain the neuritis-preventing principle, and the remaining 0·02 per cent. can be eliminated because it remained in the dialysate which failed to prevent neuritis. The 0·88 per cent. of sucrose, which is also present, is of no importance. Combining these unimportant substances and subtracting them from the 1·34 per cent. of total solids, only 0·4 per cent. remains. The neuritis-preventing substance must therefore be sought for in this 0·4 per cent. of solid matter and must be a substance that is dialysable and soluble in water, in 95 per cent. alcohol, and in 0·3 per cent. hydrochloric acid, is easily decomposed by heat and possesses a strong affinity for animal charcoal. Bodies of this description are met with among the decomposition products of proteins and attempts are now being made to identify it.—*Philippine Journ. of Science*, vi. 1911, p. 395. R. T. H.

ABSTRACT FROM 'JOURNAL OF EXPERIMENTAL MEDICINE,' Vol. xv. No. 3.

EXPERIMENTAL PRODUCTION OF LEPROSY IN THE MONKEY (*MACACUS RHESUS*), WITH A CRITICAL STUDY OF THE CULTURE EMPLOYED.

BY CHARLES W. DUVAL, M.D., AND MAURICE COURET, M.D.

(*From the Laboratories of Pathology and Bacteriology, Tulane University, New Orleans, Louisiana.*)

IN May 1911, Duval published briefly results of some experiments upon a small series of monkeys in which lesions of leprosy followed repeated injection of large quantities of acid-fast bacilli that had been cultivated from the specific lesions of human lepers. In most of the animals the lesion produced was localised at site of inoculation; but in two, lesions developed in places remote from injection site, and many of the clinical symptoms of the disease in man supervened. Even in these two monkeys, however, complete recovery apparently occurred—two to three months after onset of symptoms the animals seeming in perfect health, with disappearance of all external evidence of the infection. The experiment proved that this species of monkey could be infected with acid-fast bacilli derived from lesions of human leprosy; and seemed to establish identity of the artificially grown culture, for the experimental infection was, in many essentials, like leprosy in man. Since these earlier experiments, the authors have proved that if the animal is sensitized with killed bacilli, and given a second injection of living organisms after an interval of two or more weeks, local as well as disseminated lesions are more often induced. This is true for the monkey and for other animals hitherto regarded as refractory.

On October 17th, 1910, a full-grown *Macacus rhesus* was inoculated hypodermically into subcutaneous tissue of right inguinal region with 1 c.c. of suspension of a pure culture of acid-fast bacilli (approx. four million), isolated one year previously from the human spleen at autopsy. Eight days later approximately four billion bacilli were injected subcutaneously into left inguinal region. The monkey remained in perfect health with no local signs of reaction at inoculation sites. Two weeks after second injection a nodule, of the size of a split pea, developed in the subcutaneous tissue at the site of first inoculation; slowly increasing it attained in three weeks the size of a hazel-nut. The tumour ruptured spontaneously on the seventeenth day, the grumous discharge being found on microscopic examination to contain large numbers of acid-fast bacilli. Cultures showed no pyogenic micro-organisms.

On November 1st a third injection was made into the right groin, followed in 45 hours by the appearance of a hard nodular mass 1 cm. in extent, that slowly decreased in size.

On November 16th a fourth subcutaneous injection of the culture was given (approximately four billion bacilli), suspended in sterile normal salt solution. At this time the animal appeared in perfect health.

Three months later the animal showed the first signs of disseminated infection, and presented in part the clinical picture of human leprosy (maculo-anæsthetic type). A small raised erythematous patch, identical with the erythema noted in human leprosy, appeared on the right side of the face and slowly extended to the forehead; the expressed serum from this patch showed acid-fast bacilli in size and arrangement like the Hansen bacillus. The macular eruption persisted for two or three weeks, when it disappeared, and for nine months afterwards the monkey appeared to be in robust health with no external evidence of the infection.

Eleven months after last injection the tissues below the left eye were seen to be swollen with purplish-red overlying skin. The mass increased in size, causing complete closure of the left eye. It gave distinct fluctuation and when incised discharged 3 c.c. of thick yellow material. Under the microscope the pus showed very many slender, slightly curved, and beaded acid-fast rods, arranged in groups of twos and threes within the protoplasm of polymorphonuclear leucocytes. The gross appearance of the tumour mass and the microscopic pictures of the contents corresponded in every respect with the subcutaneous leprous abscess in the human subject. The authors point out that the pus from a human leprous abscess is composed almost entirely of polymorphonuclear cells, and is sterile with respect to pyogenic organisms (Gurd). The acid-fast bacilli in these abscesses are *B. lepræ* and not saprophytes, for the latter never give rise to acute abscess formation and are readily cultivated. The absence of acid-fast growth on all media except that for cultivation of *B. lepræ* is further proof that the culture does not contain any of the saprophytic species. The abscess was not tubercular, for subcutaneous and intraperitoneal inoculations of the pus into guinea-pigs gave negative results, macroscopically and microscopically. Eighteen days after inoculation portions of the pus were planted upon the ordinary laboratory media and special media (Dorset egg, human serum, neutral blood agar, and 1 per cent. alkaline fish agar) for the cultivation of tubercle and lepra bacilli. The ordinary media for the recovery of pyogenic bacteria remained sterile, and no growth of any kind appeared on the special media for cultivation of tubercle bacillus although incubated at 37° C. for six weeks. The alkaline fish and neutral blood agars gave a good growth of acid-fast bacilli in twelve days. The first generation showed a great variety of acid-fast rods, including many diplococcoid forms retaining intensely the carbol fuchsin stain after decolorization for one minute with Gabbet's solution. The majority of the organisms, however, were long, slender, and beaded; these had all disappeared in the third generation, nothing but deeply staining short forms being seen.

Simultaneously with the appearance of the abscess below the monkey's left eye, a subcutaneous mass developed over the external occipital protuberance, rupturing spontaneously three weeks later with discharge of serosanguineous material rich in slender, beaded, acid-fast bacilli. Microscopic sections of the hard central core of the tumour and of the thickened skin in the immediate neighbourhood showed a dense mosaic of proliferated cells of the epithelioid type interspersed with innumerable lepra-cells, many of which contained the so-called globi. The material inoculated upon a variety of media gave a growth of acid-fast bacilli only on the special lepra medium, but were associated with a staphylococcus which had gained entrance because the mass had ulcerated at the time the material was removed for cultivation. Guinea-pigs inoculated with portions of the material subcutaneously and intra-peritoneally were killed after two or three weeks respectively and showed no evidence, macroscopic or microscopic, of tuberculosis. A few weeks after the appearance in the monkey of the lesions described above several cutaneous leprous tubercles developed upon the lower part of the animal's body, one on the left leg and eight others in the right groin. The nodules were purplish red, elevated and firmer than the normal skin: several of these areas became ulcerated and the serum from them showed numbers of acid-fast bacilli of the characteristic morphology and arrangement of *Bacillus lepræ* in the human tissues. An interesting and important discovery in this case was the finding of innumerable acid-fast bacilli in the nasal secretion, although at the time of first examination there was no apparent gross naso-pharyngeal lesion. Seven days later the left external nostril had become reddened and markedly ulcerated. The animal died on December 7th, 1911, and was autopsied six hours after death. A careful and detailed examination was made, particulars of which are given, the anatomical diagnosis being: leprosy of skin (maculo-tubercular type); acute hæmorrhagic leptomeningitis (leprous); ulcerated naso-pharynx (leprous); abscess (infra-orbital). Material from the nodules in liver and spleen was removed under aseptic precautions and transplanted to the following media: Dorset egg, containing 2 per cent. glycerine, neutral blood agar, 1 per cent. tryptophan fish agar, human and rabbit serum agars, and, in addition, to the ordinary laboratory media. All remained sterile as regards micro-organisms other than an acid-fast species. The tryptophan and blood agar media showed a fine growth that was discernible to the naked eye, on the ninth day after incubation at 39° C., as minute discrete pin-point colonies. Stained preparations from these colonies showed solidly staining, short, acid-fast bacilli, resembling in morphology the bovine tubercle bacillus. The slender beaded rods of the material transplanted had changed completely under artificial cultivation. The same difference was noted with regard to the bacilli cultivated from the liver and spleen nodules. Although the organisms were morphologically and tinctorially indistinguishable in the tissue from the tubercle bacillus, their arrangement in dense clusters or packets associated with the so-called lepra-cell served at once to differentiate them. The acid-fast organism used in this experiment was isolated in pure culture from the spleen of a fatal case of human leprosy in May 1909. At the time the monkey was first inoculated the culture had been artificially cultivated for over a year, and was in consequence growing well on a variety of enriched media. At first the bacilli had multiplied very slowly, a pure culture being obtained only after repeated sub-culturing during three months upon a special medium. All the earlier attempts to colonize the culture failed, though it grew readily upon nutrient alkaline agar in association with amœba and *B. typhosus*; however, after repeated sub-planting, separate colonies were obtained after five or six weeks' incubation at 32° C.

Had the transplanted human leprous tissue contained some common saprophytic acid-fast species, in addition to the Hansen bacillus, no difficulty would have been encountered in its cultivation, nor would it have taken months to accomplish its isolation. The ordinary saprophytic species, which might be confused with *Bacillus lepræ*, grow readily upon almost any laboratory medium. The source of this culture (spleen) makes it improbable that it is an extraneous acid-resisting bacterium such as the bacillus of Timothy hay, &c., even granting that some of the so-called confusing group may accidentally occur at times in the exposed leprous ulcer.

In general, the lepra culture retained for many generations the morphology of the Hansen bacilli in the tissues. After the twentieth or thirtieth generation on artificial medium, at which period artificial growth was permanently established, the bacilli showed decided change in size, shape, and arrangement. Diplobacillary forms appeared resembling the young cultures of bovine tubercle bacillus. The difference was so marked that, unless the transformation had been followed from day to day, one would doubt whether the resulting growth of small, deeply staining rods was the progeny of the original beaded forms. It was repeatedly observed that this same culture when passed through a susceptible animal again assumes the characteristic slender beaded appearance of the Hansen bacillus of the tissues. Although this transformation indicates that the culture is rapidly growing and has become accustomed to saprophytic conditions, it is no criterion that the organism is incapable of producing leprosy, as the experiment reported herein proves.

The change in morphology of the bacilli in culture is easily followed by a routine microscopic study. Massing of chromatin takes place in the individual bacilli to form one or more swollen ovoid bodies indicating a mitotic division: this becomes more definite until one or two well formed deeply staining oval bodies appear in the parent cell; eventually the culture consists entirely of single and diplobacillary forms. There is hardly a possibility for those experienced in the initial cultivation of the *B. lepræ* from the tissues to take the two forms as distinct species, or to assume that the long variety is the true Hansen bacillus which failed to multiply and in consequence became lost or killed by the other species. Apart from the fact that the diplococcoid form of *B. lepræ* is capable of producing leprous lesions and will again revert in the animal body to the basic type (slender and beaded), there are distinct biological differences that serve to separate the culture from all known saprophytic acid-fast species. In each instance where the authors have attempted the cultivation of the Hansen bacillus, the resulting growths have corresponded in every particular. The probability of always recovering the same saprophyte from internal as well as external lesions in every case of leprosy from which they had cultivated an acid-fast bacillus seems extremely remote. Some strains of *B. lepræ* are recovered in pure culture more easily than others: but in most instances the specific organism is recovered with great difficulty, and only after making sub-cultures on special medium. Again, in a few cases it is almost impossible to obtain *B. lepræ* in pure culture, though growing well in symbiosis with *B. typhosus*, &c.

The initial culture of *B. lepræ* cannot be confused with any of the common acid-fast saprophytes, though old cultures that have become accustomed to artificial growth conditions and are multiplying readily may possibly be confused with the bacillus of Timothy hay, because both produce a somewhat similar pigment. Even with regard to this property a comparative study shows distinct and constant differences for the two species. There is, however, no chance of mistaking *B. lepræ* for any of the other known acid-resisting saprophytes, such as Moeller's grass bacilli and Rabinowitch's cultures from butter and milk.

Pure cultures of *Bacillus lepræ*, grown upon solid medium, especially on 1 per cent. alkaline fish agar, always give a moist cadmium-coloured growth, glistening, easily detached, and not spreading from needle track; while the Timothy hay bacillus, the only species with which *B. lepræ* might be confused, invariably produces a dull, dry, brick-red growth, rapidly spreading over the whole surface of the medium. On the most favourable medium the most rapidly growing cultures of *B. lepræ* take three to five days to show visible growth, while the Timothy hay bacillus, under the same conditions, gives a well-marked growth in twenty-four hours. The latter grows profusely on ordinary nutrient agar, while *B. lepræ* either fails to grow or at most gives a very faint growth on that medium. *B. lepræ*, again, is a facultative ærobe, while all strains of the Timothy hay bacillus with which the authors have worked are obligate ærobes. *B. lepræ* is readily emulsified, Timothy hay bacillus only with difficulty, like *B. tuberculosis,* or not at all. On glycerine bouillon (Theobald Smith's) the end reaction for *B. lepræ* is neutral or slightly alkaline, the bacillus of Timothy hay producing no change in the reaction of the medium. Finally, serological tests are specific and serve to differentiate sharply the lepra culture from any of the known saprophytic acid-resisting group.

The authors conclude:—Fatal leprosy, with all its clinical and pathological manifestations in man, may be experimentally induced in the monkey (*Macacus rhesus*) with a pure culture of the acid-fast bacillus, cultivated by Duval from a leprous lesion in man.

To produce the disease experimentally, repeated injections of large numbers of the bacilli must be made at given intervals for a period of months.

Infection is more likely to follow where sensitization is first established. It is assumed that the first injection sensitizes the animal; and it may consist of either killed or viable lepra bacilli. The necessity of first sensitizing the monkey and then giving repeated doses of viable organisms over a long period offers perhaps an explanation of the fact that man rarely, if ever, contracts leprosy when intimately associated for an indefinite period with those afflicted with the disease. The leprous lesions in the monkey are histologically indistinguishable from those in man, and do not essentially resemble the specific lesion of tuberculosis or blastomycosis. The appearance of large lepra-cells and the arrangement of the bacilli in dense packets within these cells to form the so-called globi is a constant and characteristic feature for the experimental, as well as the human, lesion.

It is no indication that a given culture is not the Hansen bacillus because the individual organisms differ in size and shape from those in the tissues, since it is a well-known fact that marked variations in morphology are common for many bacterial species under natural and artificial conditions. Couret has pointed out that there is wide variation in the morphology of *B. lepræ* under different environment. The experimental work emphasizes this fact and is a proof that the slender beaded rods of *Bacillus lepræ* do become transformed into solidly staining diplococcoid forms in culture, and these again may assume the slender beaded appearance by passage through warm-blooded animals.

The interest and value of the paper are much increased by the appended photomicrographs and coloured plate illustrating clearly the essential points discussed. H. M. H.

ABSTRACT FROM 'BRITISH JOURNAL OF DERMATOLOGY,' April 1912.

TRICHOSPOROSIS NODOSA.

By J. M. H. MACLEOD, M.D.

THE specimens of hair which form the subject of this paper were obtained from Dr. E. Minett, Government Bacteriological Department, British Guiana, and were hairs from an aboriginal Indian girl, of the tribe Atavy, living in the Rupununi District.

The hairs were black, rather fine, and on the shaft were a number of hard nodules varying in size from minute specks like particles of grit up to small pins' heads. In some cases nodules invisible to the naked eye could invariably be felt. The nodules, roughly oval in shape, brownish black in colour, hard and firmly attached, were situated either on the side of the hair-shaft or formed a concretion completely ensheathing it. They were distributed irregularly on the shaft and varied in number from two or three to twelve or more. They were most numerous at some distance from the scalp, and were stated to be absent on the intra-follicular portion of the hair.

Microscopical appearances.—When softened in liquor potassæ and examined under a low power of the microscope, the nodules were found to consist of a mass of spores forming a concretion, at first entirely outside the hair cuticle. Later, as concretions increased in size, they appeared to destroy the cuticle and even cortex, separating the cells, causing the hair surface to become frayed and rendering it liable to fracture opposite them. With a higher power the mass appeared made up almost entirely of spores, no definite mycelial filaments being detected. These spores were usually large and were recognised as being most probably those of the fungus to which Behrend gave the name of *Trichosporon giganteum*. These had a strikingly regular arrangement. At the periphery of the nodule they were oblong in shape, about 12 μ in length and 6 μ in breadth, and were arranged at right angles to the hair-shaft, forming a palisade layer, while next the hair they were irregular and polygonal in outline and tended to be arranged like the cells of a honeycomb or mosaic. Sometimes there was a peculiar fan-like grouping with rows of spores radiating from a central base. The spores had a yellowish appearance owing to the presence of diffuse colouring matter and pigment granules in the protoplasm, and were held together in a mass by a viscid cement substance which formed a sort of capsule enclosing them.

Cultivation.—With the assistance of Mr. W. Topley, Bacteriologist, Charing Cross Hospital, a characteristic culture was eventually obtained by soaking small pieces of the hair containing nodules for 5 minutes in absolute alcohol and planting them on proof agar. Nodules grown in broth gave a white flocculent culture, which when planted on proof agar gave the same growth as that obtained directly. The agar tubes were incubated at about 16° C.

About one week after inoculation a small white solid speck, about the size of a large pin's head, appeared on the surface of the medium; this increased in the course of a week to the size of a split pea and assumed a greyish-green tint, which spread from the centre to the periphery and had a slightly powdery surface but no marked duvet. One week later the culture was as large as a sixpence, forming a heaped-up roundish mass with crenated nodular border, and the greyish tinge had become replaced by a brownish colour which gradually darkened to that of milk chocolate. Microscopical preparations from the culture in broth showed irregular branching mycelial filaments, with spores at varying intervals and endogonidia containing small spores of different sizes, and granules. The cultural appearances corresponded with those which were obtained by Minett.

General remarks.—There is little doubt that the condition described is identical with that known, on account of its stony hardness, as "Piedra" in Columbia. It has been observed also in India and Ceylon (Castellani). A closely allied condition has been known for many years in Europe and has been described under a variety of names: *Tinea nodosa* (Morris & Cheadle), *Piedra nostras* (Behrend & Trachsler), *Trichomycosis nodularis* (Dubel-Penoy), and in all probability it was the "chignon fungus" of Berjel and Tilbury Fox.

In the Tropics it generally affects the scalp hairs of native women, but it has been found also on the eyelashes, hair, moustache, and beard in men; in most of the cases in the temperate zone it has affected the beard or moustache. The tropical cases are usually due to a large spored fungus of the type *Trichosporon giganteum* (Behrend). According to Castellani, the Ceylon cases are caused by a fungus somewhat different from it. The varying descriptions of the fungi seen in the European cases point to the conclusion that the condition may result from a number of allied fungi. The origin and means by which the fungus is implanted on the hairs is uncertain. It is known to be contagious.

Diagnosis.—Trichosporosis nodosa has to be distinguished from the more common affection known as leptothrix. Confusion has arisen between these two quite distinct conditions on account of certain names which have been given to leptothrix, such as trichomycosis nodosa (Patterson), trichomycosis palmellina, and trichomycosis tropica (Castellani). In leptothrix the hairs of the axilla and occasionally the scrotum are involved; it is much more common than trichosporosis nodosa. In the axillæ it is often associated with red sweat. In leptothrix the hairs, instead of having a nodose appearance, are thick, irregular, and ragged, owing to the presence of concretions on the surface which are broken up into lobes arranged roughly like the barbs of a feather, or form irregular reddish-brown masses. The concretion eventually affects the hair itself, causing splitting up of the fibres and a tendency to fracture in a brush-like manner. These concretions, instead of being made up of spores, appear to be chiefly composed of a cement substance, which becomes hard and chitinous and is allied to that which fixes nits to the hair-shaft. This cement is believed to be secreted by a micro-organism, and various microbes have been described in connection with it; but so far none has been definitely established as the cause.

Castellani observed it in Ceylon, where it affects the axillæ and presents three varieties, which he has named trichomycosis flava, nigra, and rubra; the yellow and black varieties being the commoner. According to him, the yellow is due to a bacillary-like fungus, probably a streptothrix or a microsporoides, which he was unable to cultivate. The yellow and black pigment he attributes to coccus-like organisms which grow on the hair in symbiosis with the streptothrix. The black pigment-producing coccus is easily cultivated, and he suggested the name *Micrococcus nigrescens* for it. The red pigment-producing coccus somewhat resembled the *Micrococcus ruber* of Tromsdorff, found in cases of chromodrosis.

Treatment of trichosporosis nidosa is unsatisfactory, chiefly on account of the practical difficulty of removing the concretions from the hair, and the condition never showing any tendency to spontaneous recovery,

The treatment indicated is similar to that employed for the removal of nits from the hair. This, however, seldom effects a cure, and it is generally necessary to shave the scalp or cut the hair short: antiseptic precautions, reapplied daily, must be taken to prevent any of the spores which may be left about growing on to the hairs again.

The paper is well illustrated with drawings and photo-micrographs of the fungus in culture and on the hair, clearly showing its structure.

H. M. H.

V.—PUBLIC HEALTH.

By Prof. R. T. HEWLETT, M.D.

A Cheap and Simple Process for the combined Clarification, Decolorisation, and Sterilisation of Peaty Waters.

By K. S. WISE, M.B., B.S., B.Sc. (Lond.), D.P.H.,
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and
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Assistant Government Bacteriologist, British Guiana.

The usual source of drinking-water in this Colony is rain-water collected from the roofs of dwelling and other houses, and stored usually in vats and tanks. These receptacles are constructed of either wood, iron, or stone, and the average annual rainfall being approximately 100 inches there is usually no difficulty in obtaining good supplies.

But early in 1912, owing to a prolonged drought, the city was faced with a water famine, there being no rain-water available; the few artesian wells scattered over the city were either useless through long disuse or were too brackish to suit the taste of a people accustomed to rain-water.

The wells usually contained a large amount of iron and magnesium salts, and the chlorides expressed as Cl_2 ranged from 26 up to 105 parts per 100,000.

In addition to the two foregoing sources of water the city possesses a pipe-borne supply of brown peaty water, so-called Lamaha water, drawn from a service canal running into the town. This water varies from a pale sherry to a dark brown colour; it contains varying quantities of peaty organic material in solution and in suspension, usually about 4 parts in 70,000, together with iron in varying quantities in the ferrous state. It is free from sewage in the upper reaches of the service canal, fæcal bacilli being absent from 10 c.c. After having passed through the service pipes of the city to the consumers it is very impure and badly contaminated, fæcal bacilli being frequently present to the extent of 10 per c.c. The following are analyses of a few samples taken at random from the pipe water supply:—

No. of sample.	Total organisms per c.c.	Fæcal bacilli per c.c.	Streptococci.
1.	2400	20	Present 5 c.c.
2.	154	10	....
3.	3800	Present 1 c.c.	Absent 5 c.c.
4.	8160	Present 1 c.c.	Absent 5 c.c.
5.	2600	10	Present 5 c.c.
6.	1640	Present 1 c.c.	Absent 5 c.c.

As will be seen, this is anything but a desirable water-supply as it reaches the consumer, however pure it may have been originally. The pipes passing through the sewage-saturated ground in many parts of the city are extremely liable to allow of the entry of sewage pollution by either a defective joint or negative pressure.

As this peaty water is very soft and very plumbo-solvent, it also takes up additional iron from its passage through the pipes.

Early in 1912, owing to the drought, the rain-water supplies in the city were exhausted and it became necessary to utilize the peaty impure Lamaha water for drinking purposes. After a long series of experiments with various chemicals in the laboratory, including perchloride of iron, lime, manganese, alumino-ferric cake, animal charcoal, clay, sand, &c., it was finally decided to use a sedimentation process, using alum as a precipitant and chlorinated lime as a bactericide. The usual quantity of alum added was 4·8 grains per gallon, but in some cases where the water was deeply coloured it was found necessary to increase this to 5·25 grains per gallon. Chlorinated lime was added in the proportion of ·48 grain per gallon with a double object: firstly, to sterilise the water by means of the 2 parts per 1,000,000 of free chlorine available from this quantity of the chlorinated lime; secondly, to assist the total precipitation of the alum as aluminum hydrate. This process was found to be eminently successful in producing a perfectly clear, colourless, and palatable drinking-water. The various empty vats, tanks, cisterns, &c. for storing rain-water throughout the city were filled with Lamaha water and treated *in situ* by this process; in all, about 10,000,000 gallons of water were treated and used for drinking purposes.

The following are chemical and bacteriological analyses of a bad sample of this water before and after treatment by this process.

Sample of water before treatment:—

CHEMICAL ANALYSIS.

Physical characters:—

Turbidity moderate. Colour pale yellowish brown. Odour not marked. Reaction faintly acid.

Ammonia (free)	·06	parts per	100,000
Ammonia (albuminoid)	·15	,,	,,
Chlorine	2·3	,,	,,
Nitrites	Nil.		
Nitrates	Nil.		
Hardness (total)	3·2	,,	,,
Solids (total)	28·0	,,	,,
,, Organic	15·2	,,	,,
,, Inorganic	12·8	,,	,,
Phosphates as P_2O_5	·3	,,	,,

Microscopic examination of deposit:—

Many small ciliata present. Larvæ of ticks. Cotton fibres, &c.

BACTERIOLOGICAL ANALYSIS.

Total organisms capable of growing on agar at 37° C.	4800 per c.c.
Bacillus Coli Communis	200 per c.c.
Bacillus Enteriditis Sporogenes	Not investigated.
Streptococcus Fæcalis	Present in 1 c.c.

Other organisms present: Bacillus proteus vulgaris.

The same water after treatment :—

CHEMICAL ANALYSIS.

Physical characters :—

Turbidity nil. Colour nil. Odour nil.
Reaction faintly alkaline to litmus.

*Ammonia (free)	2·4	parts per	100,000
Ammonia (albuminoid)	·08	,,	,,
Chlorine......................	3·3	,,	,,
Nitrates	Nil.		
Nitrites	Nil.		
Hardness (total):			
(*a*) With sediment	12·0	,,	,,
(*b*) Clear fluid	8·0	,,	,,
Phosphates as P_2O_5	Traces only.		
Solids (total)....................	8·0	,,	,,
,, Organic	2·5	,,	,,
,, Inorganic	5·5	,,	,,

Microscopic examination of sediment nil, as the sample was taken from supernatant clear water and practically no deposit settled afterwards in bottle.

* High figure partly due to use of ammonia alum as precipitant.

BACTERIOLOGICAL EXAMINATION.

Total organisms capable of growing on agar at 37° C.	96 per c.c.
Bacillus Coli Communis................................	Absent from 10 c.c.
Bacillus Enteriditis Sporogenes	Absent from 100 c.c.
Streptococcus Fæcalis................................	

Note.—Analyses as above were taken two hours after addition of alum and half an hour after addition of chlorinated lime.

As will be seen by the foregoing analyses, the results of the process were eminently satisfactory from the point of view of a potable water of guaranteed purity. The only drawback was that the water even after purification was still both plumbo- and ferro-solvent to a high degree, so that were it proposed to carry this water long distances through pipes the further addition of lime would be necessary in order to neutralize the acidity of the acid salt of aluminum added. No free acid could be detected by means of Congo red paper, the acidity being apparently due entirely to the acid salt.

The amount of solids present in the water varies slightly, but the following is fairly typical :—Alum 5 grains was added to 1 gallon of the peaty water and allowed to stand 24 hours. The precipitate was then collected on a filter-paper, dried in a hot-air oven, and weighed. The total precipitate was 9 grains ; this after heating to redness weighed 5·1 grains. Thus, for 1 gallon of water with 5 grains alum added,

Total solids, 9 grains : organic solids, 3·9 grains ; inorganic solids, 5·1 grains.

The deposit after incineration contained iron, alumina, calcium, sodium, potassium, and silica.

The supernatant clear fluid after evaporation to dryness weighed 4·35 grains; after incineration it weighed 3·4 grains. The latter consisted of iron, alumina, magnesium, calcium, sodium, and potassium, a considerable quantity of phosphates being present.

Alum alone cannot be depended upon as a bactericide in the peaty waters. It forms a flocculent precipitate which carries down many of the bacteria mechanically but does not destroy them. It is not a germicide, as the following experiment shows :—

Lamaha water infected with fæces and treated with alum only. The same sample is used throughout the experiment, and samples are analyzed at frequent intervals.

Lab. No.	Date.	B.C.C. per c.c.	Remarks.
4486	18.1.12	1000	Before treatment with alum.
4496	23.1.12	Absent from 3 c.c. Present in 6 c.c.	After addition of alum only.
4508	24.1.12	Absent 1 c.c. Present 2 c.c.	Clear supernatant fluid.
4525	25.1.12	Absent 1 c.c. Present 2 c.c.	Clear supernatant fluid.
4537	26.1.12	Absent 4 c.c. Present 6 c.c.	Clear supernatant fluid.
4553	27.1.12	Present 1 c c.	Clear supernatant fluid.
4585	30.1.12	Absent 6 c.c.	Clear supernatant fluid.
4603	31.1.12	Absent 6 c.c.	Clear supernatant fluid.
4610	1.2.12	Present 2 c.c.	After stirring up deposit.
4618	2.2.12	Present 1 c.c.	After stirring up deposit.
4630	3.2.12	Absent 1 c.c. Present 3 c.c.	Clear supernatant fluid.
4639	5.2.12	Absent 1 c.c. Present 2 c.c.	Clear supernatant fluid.
4660	6.2.12	Present 1 c.c.	After stirring up deposit.
4669	7.2.12	Absent 6 c.c.	Clear fluid.
4677	8.2.12	Absent 6 c.c.	Clear fluid.
4682	9.2.12	Present 1 c.c.	After stirring up deposit.

Note.—The clear water is never or very seldom wholly free from B.C.C., although the majority are carried down by the precipitate. But a large number of B.C.C. reappear in the water immediately the deposit is stirred up, although no further addition of fæcal material had been made.

As will be seen by the foregoing experiment, the addition of alum alone to the water is not sufficient to ensure its purity. Large numbers of fæcal organisms are undoubtedly carried down by the dense flocculent precipitate formed, but they are not destroyed ; they reappear when this deposit is disturbed, and even, as we have proved by subsequent experiments, can grow through the deposit and reappear even in the surface layers of the supernatant fluid.

The following experiment was carried out with a view to ascertaining whether the water once purified will remain pure provided no further pollution is added, it being considered unnecessary to prolong the experiment over 21 days in order to demonstrate this point. As will be seen by reference to the first three analyses, the sterilisation was not complete until after addition of the chlorinated lime (chlorine), the mechanical purification of the aluminum precipitate being unable to deal effectually with all the bacteria present, although making a considerable reduction in their numbers.

Purified water will remain sterile if no further pollution is added.

Experiment.—A quantity of Lamaha water was infected with fæces 1 grm. per gallon and allowed to stand 24 hours.

The same sample is used throughout and analyses made at frequent intervals.

Time of Analysis.	Lab. No.	Date.	Total organisms per c.c.	B.C.C. per c.c.
Before treatment	4163	15.12.11	2816	100
After alum treatment	4165	16.12.11	247	Present 2 c.c.
After chlorine treatment (4 hours) . .	4168	16.12.11	Nil.	Absent 21 c.c.
,, ,, (24 hours) . .	4169	17.12.11	Nil.	,,
,, ,, (48 hours) . .	4172	18.12.11	15	,,
,, ,, 3 days	4202	19.12.11	Nil.	,,
,, ,, 4 days	4214	20.12.11	Fair numbers of very small colonies.	,,
,, ,, 5 days	4216	21.12.11	,,	,,
,, ,, 6 days	4223	22.12.11	4 colonies only.	,,
,, ,, 7 days	4234	23.12.11	Not done.	,,
,, ,, 8 days	4235	24.12.11	,,	,,
,, ,, 9 days	4236	25.12.11	,,	,,
,, ,, 10 days	4237	26.12.11	,,	,,
,, ,, 11 days* . .	4245	27.12.11	,,	,,
,, ,, 12 days* . .	4280	28.12.11	,,	,,
,, ,, 14 days* . .	4293	30.12.11	,,	,,
,, ,, 21 days* . .	4335	6.1.12	,,	,,

* On these occasions the sediment remaining at the bottom of the tank was stirred up in order to demonstrate that the sterilisation was not merely mechanical.

The next experiment was carried out in order to demonstrate that, up to a certain point, water once sterilised by the addition of 2 parts per 100.000

of chlorine can deal with a fair amount of subsequent pollution and still remain a good potable water.

Lamaha water after precipitation and sterilisation can deal with successive additions of fæcal material up to a certain point.

Experiment.—A quantity of Lamaha water was infected with fæces 1 grm. per gallon and allowed to stand 24 hours at room temperature.

Results of Analysis on same water.

Lab. No.	Date.	Total organisms per c.c.	B.C.C. per c.c.	Remarks.
4351	9.1.12	489,600	10,000	B. violaceus present.
4361	10.1.12	48,000	1,000	B. violaceus present, alum only added.
4367	11.1.12	Sterile.	Absent 6 c.c.	1 hr. after addition of chlor. lime.
4373	11.1.12	50	,,	After further addition of fæces.
4396	12.1.12	Not done.	Absent 2 c.c. Present 3 c.c.	More fæces added.
4398	13.1.12	,,	Absent 6 c.c.	To confirm 4396, no fæces added.
4407	15.1.12	,,	,,	After further addition of fæces.
4424	16.1.12	,,	Present 1 c.c.	To confirm 4407.
4425	16.1.12	,,	Present 2 c.c. Absent 1 c.c.	16 hrs. after further addition of fæces.
4459	19.1.12	,,	Absent 6 c.c.	No further addition of fæces.
4467	20.1.12	,,	,,	To confirm 4459.
4483	22.1.12	,,	Present 1 c.c.	After further addition of fæces.
4495	23.1.12	,,	Absent 3 c.c. Present 6 c.c.	,, ,, ,,
4507	24.1.12	,,	Absent 4 c.c. Present 6 c.c.	To confirm 4495.
4520	25.1.12	,,	Present 1 c.c.	To confirm 4507.

Note.—The deposit at bottom of vessel was stirred up after each fresh addition of fæcal material and again before taking samples for analysis.

As will be seen, the quantity of chlorine added was sufficient to sterilise an exceedingly foul water in one hour, and, further, that after six successive additions of fæcal material the water was fairly good after 15 days of continuous pollution. This we attribute to the fact that two parts Cl_2 per 100,000 leaves a margin of somewhere about 1·4 parts Cl_2 per 100,000 free and available to deal with successive pollutions. This lowest limit is put at ·6 parts per 100,000 by Thresh, working with B. typhosus as his organism; this is further borne out by the following experiment, designed as a check on the combined process and to prove that chlorine added to polluted water is at once efficient, and, further, that the amount of chlorine added (two parts per 1,000,000) allows a generous margin of safety.

Experiment.—Lamaha water infected with fæces 1 grm. to 1 gallon and allowed to stand 24 hours at room temperature.

Same sample throughout. Chlorinated lime only added.

Lab. No.	Date.	B.C.C. per 1 c.c.	Remarks.
4487	20.1.12	1000	Before addition of chlorine.
4497	23.1.12	Absent 6 c.c.	½ hour after addition of chlorine.
4509	24.1.12	,,	After further addition of fæces.
4536	26.1.12	,,	No further addition of fæces.
4553	27.1.12	,,	,, ,, ,,
4586	30.1.12	,,	From clear supernatant fluid.
4604	31.1.12	,,	,, ,, ,,
4611	1.2.12	,,	After stirring up deposit.
4619	2.2.12	,,	From clear fluid.
4631	3.2.12	,,	,, ,,
4640	5.2.12	,,	,, ,,
4661	6.2.12	,,	,, ,,
4670	7.2.12	,,	,, ,,
4678	8.2.12	,,	,, ,,
4682	9.2.12	,,	After stirring up deposit.

Note.—Although the deposit was repeatedly stirred up the water remained free from B.C.C. Chlorine is therefore a true germicide and does not act mechanically like alum.

This process has served well as a cheap and efficient method to use as a temporary measure, such as was necessary in British Guiana during the dry period of this year. We are of course aware that many other substances can be used as precipitants for peaty water—sulphate of alumina, for example; but when suddenly called upon to treat large quantities of water, this was unobtainable in the Colony, so we used the cheapest materials to hand. The use of chlorine is of course also well known as a disinfectant, and we claim nothing original for its use beyond the fact that by using chlorinated lime containing roughly 30 per cent. of available chlorine instead of slaked lime to precipitate the excess of alumina we served the double purpose of providing a precipitant and an efficient bactericide at the same time.

Ferric chloride and lime is sometimes used as a means of clarifying peaty water in this colony; but after many experiments with this process we discarded it for the following reasons:—

That it is inefficient unless a large excess of alkali is added, and, further that the exact quantity of iron, with excess of alkali, must be used or else the small quantity of iron remaining in solution imparts a very unpleasant "inky" taste to the water. Carried out with accuracy it gives an excellent result ; but unfortunately this is impossible where natives are concerned.

Various oxidising agents are very successful, such as permanganate of potash, but they all require a further filtration through charcoal and sand to produce a satisfactory result, thus unduly complicating the process.

The alum and chloride of lime method although eminently successful as a precipitation method when employed in large tanks, vats, &c., was not in our hands successful as a continuous process used in conjunction with sand filtration. It was found that the precipitate formed, rapidly clogged the filters, rendering them useless.

We therefore, after a lengthy trial of a combined precipitation and filtration process, reverted to a precipitation process only as giving the best and most satisfactory results.

We beg to acknowledge our indebtedness to Professor Harrison and Dr. Thresh for kind assistance and advice.

JAIL DIETARIES OF THE UNITED PROVINCES.—Major McCay, I.M.S., has made an elaborate investigation of the nutritive value of the diets at present in use in the jails of the United Provinces, and has worked out the coefficients of protein and carbohydrate absorptions of the different food-materials entering into those dietaries. From the data obtained new dietaries have been framed, the nutritive value of which depends on the degree of nitrogen absorption possible from the food materials and not on their gross chemical composition. A series of eight such diets are given, which are practically of identical nutritive values and are therefore interchangeable. The loss of protein by the fæces is very great when inferior food-stuffs are used, for the percentage of nitrogen in the fæces remains practically constant whatever the type of diet may be. From the data obtained it would appear that, other things being equal, diet is the all-important factor in determining the degree of physical development and general well-being of a people, and that with a low level of nitrogenous interchange deficient stamina, morally and physically, must be expected.—*Scientific Mem. of the Gov. of India*, No. 48. R. T. H.

VI.—CLINICAL MEDICINE.

FURTHER REPORT ON THE NASTIN TREATMENT OF LEPROSY CARRIED OUT AT THE LEPER ASYLUM, MAHAICA, BRITISH GUIANA, FROM SEPTEMBER 1910 TO SEPTEMBER 1911.

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PART I.

SINCE September 1910 this treatment has been carried out on the same lines as before, a few minor modifications only being introduced.

Of the 24 cases selected 18 were under treatment with nastin for practically two years, the remaining 6 cases were under treatment for periods ranging from six to nine months.

In September 1910 all these cases were transferred from treatment with nastin only to treatment with a solution of benzoyl chloride.

Results of the 24 cases treated first with nastin and later with benzoyl chloride:—

Males	19
Females	5
	24

Race.

	Males.	Females.	
Black	12	4	
E. Indian	2	0	
Coloured	2	1	
Portuguese	1	0	
White	2	0	
	19	5	Total 24.

Country of Origin.

	Males.	Females.	
British Guiana	14	5	
Barbadoes	3	0	
India	2	0	
	19	5	Total 24.

Age Incidence.

	Males.	Females.	
10 to 20 years	7	3	
20 to 30 „	5	2	
30 to 40 „	7	0	
	19	5	Total 24.

Approximate Duration of Disease when Treatment started.

	Males.	Females.	
1 year and under	9	3	
1 to 3 years	5	1	
3 years and over	5	1	
	19	5	Total 24.

Duration of Treatment.

	N.		B. C.		
	M.	F.	M.	F.	
Under 1 year	6	0	9	3	
1 year and over	13	5	10	2	
	19	5	19	5	Totals 24.

Presence or Absence of Bacilli, with Estimation of Numbers Present at End of Period of Observation.

	Males.	Females.	
Many Bacilli	10	3	
Few Bacilli	5	1	
No Bacilli	4	1	
	19	5	Total 24.

Amount of Destruction of Bacilli observed.

	Males.	Females.	
No destruction	3	0	
75 % „	7	0	
50 % „	2	1	
25 % „	3	3	
No bacilli	4	1	
	19	5	Total 24.

Type of Cases.

	Males.	Females.	
Nodular	9	2	
Anæsthetic	4	3	
Mixed	6	0	
	19	5	Total 24.

Presence or Absence of Ulcers.

	Present.	Absent.	
Males	13	6	
Females	3	2	
	16	8	Total 24.

Cases in which Eye-Lesions were Present.

	Present.	Absent.	
Males	1	18	
Females	1	4	
	2	22	Total 24.

Classification of Results after Treatment.

	Males.	Females.	
Improved	4	1	
Doubtful	8	2	
Worse	7	2	
Died	0	0	
	19	5	Total 24.

Careful clinical notes were taken at regular intervals of the condition of the patients under treatment.

A microscopical examination was also made frequently and the findings noted under the date when the specimens were taken.

The numbers of bacilli present were estimated by the general characters of each slide in conjunction with the numbers of bacilli observed in several fields. As the whole of the estimations were made by the same observer they may, I think, be taken to be approximately correct.

The amount of so-called destruction of bacilli was estimated by observing the number of bacilli showing plasmolysis in a constant total, the estimation being the work of the same observer in all cases in order to ensure uniformity of results.

The results of this treatment will be discussed later in the report.

PART II.

Report on 71 Cases treated with Benzoyl Chloride.

No attempt was made to select these cases, the patients being taken under treatment as they presented themselves.

The treatment consisted in intramuscular injections of a 5 per cent. solution of benzoyl chloride in sterilised heavy petroleum oil (white oleum petrolatum). This was used in preference to an animal or vegetable oil owing to its freedom from water, as many of the former contain a considerable amount of water and cause a rapid decomposition of the benzoyl chloride.

This decomposition does not take place in a mineral oil, which also has the additional advantage in that it does not become rancid if exposed to the air.

In addition to the injections a 2½ per cent. solution of benzoyl chloride in oleum petrolatum was used weekly as a nasal spray, and ulcers and broken-down nodules were painted with this preparation.

The following are the results of these 71 cases in tabulated form :—

Total number of Cases treated 71.

Sex Distribution.

Males	46
Females	25
	71

Country of Origin.

	Males.	Females.	
British Guiana	22	18	
India	20	4	
Barbadoes	3	2	
Africa	1	0	
Madeira	0	1	
	46	25	Total 71.

Race Incidence.

	Males.	Females.	
Black	19	15	
East Indian	23	5	
Mixed	2	1	
White	0	1	
Portuguese	2	3	
	46	25	Total 71.

Age Incidence.

	Males.	Females.	
10 to 20 years	8	5	
20 to 30 ,,	12	7	
30 to 40 ,,	13	5	
40 to 60 ,,	13	8	
	46	25	Total 71.

Approximate Duration of the Disease when first treated.

	Males.	Females.	
Under 6 months	13	10	
6 months to 1 year	12	6	
1 year to 3 years	9	3	
3 years to 5 years	4	1	
Over 5 years	8	5	
	46	25	Total 71.

Length of Treatment.

	Males.	Females.	
Under 6 months	1	2	
1 year and over	45	23	
	46	25	Total 71.

Presence or Absence of Bacilli (at end of experiment).

	Males.	Females.	
Many bacilli	20	6	
Few bacilli	5	6	
No bacilli	16	10	
Irregular cases	5	3	
	46	25	Total 71.

Destruction of Bacilli observed.

	Males.	Females.	
No destruction	2	1	
75 % ,,	10	1	
50 % ,,	5	3	
25 % ,,	5	4	
No bacilli present	24	16	
	46	25	Total 71.

Type of Cases.

	Males.	Females.	
Nodular	16	9	
Anæsthetic	25	14	
Mixed	5	2	
	46	25	Total 71.

Presence or Absence of Ulcers.

	Males.	Females.	
Present	26	16	
Absent	20	9	
	46	25	Total 71.

Presence of Eye-Lesions.

Males	2
Females	3
Total	5 Cases.

Classification of Results.

	Males.	Females.	
Improved	18	7	
Worse	9	4	
Doubtful	19	11	
Died	0	3	
	46	25	Total 71.

Comparison between 24 cases treated with nastin, 71 cases treated with benzoyl chloride, and 8 cases that had received no treatment of any description, beyond removal to better conditions as regards food, cleanliness, &c., as obtained in the Asylum in comparison with their former conditions of poverty :—

Expressed as percentages of each total.

	Nastin. per cent.	Benzoyl Chloride. per cent.
Improved	21	36
Doubtful	42	42
Worse	37	19
Died	0	3
Bacilli still present	79	52
Ulcers present	41	59
Eye-lesions	8	7

PART III.

Eight untreated cases were examined regularly as confirmatory evidence that anæsthetic leprosy tends to self cure; the following shows briefly their classification :—

Patients considered as non-infective that have never been treated in any way.

Total number...... 8. All males.

Country of Origin.

British Guiana	7
India	1
Total	8

Duration of Disease.

5 years	1
5 to 10 years	3
10 to 20 years	4
Total	8

Race Incidence.

Black	7
East Indian	1
Total	8

Age Incidence.

20 to 30 years	2
30 to 40 ,,	2
40 to 50 ,,	2
50 to 60 ,,	2
Total	8

Type of Disease.

Anæsthetic	6
Mixed	2
Nodular	0
Total	8

All show continued absence of bacilli.

Two show no ulcers, 6 have small ulcers.

Six show signs of decided improvement. Although contractions and amputations are present they appear no longer infective.

With regard to the plasmolysis or so-called destruction of bacilli, it has been before stated that this is a natural process and can be observed in untreated lepers. There appears to be no doubt that this view is a correct one, as after making careful notes of many cases in various stages of the disease, all of them untreated, it can be shown that the degree of destruction is totally unreliable as an index of the progress of treatment. Apparently the damaged appearance of the bacilli is merely the effect on them of a local tissue reaction, probably a partial phagocytosis or a bacteriolysis ; it varies from time to time and also in different situations in the same patient.

The following are the results of twelve cases examined periodically before any treatment was started. They were recorded for the purpose of having control cases to compare with the amount of destruction observed in the cases treated with nastin and benzoyl chloride. The amount of destruction was estimated in an exactly similar manner in all cases.

PART IV.

Untreated Cases.

Analysis of 12 cases examined before treatment of any kind was commenced.

Presence or Absence of Bacilli.

Many bacilli	8
Few bacilli	4
Total	12

Amount of Destruction of Bacilli observed.

75 % destruction	5
50 % ,,	2
25 % ,,	3
Destruction not estimated	2
Total	12

GENERAL REMARKS.

As regards the clinical observations the general effect was that the patients seemed brighter, and the Medical Superintendent stated that they worked with a better will. This effect was equally well marked whether the cases were treated with nastin or benzoyl chloride.

I am of opinion that the improvement is almost entirely due to their improved mental condition ; but whether entirely so it is difficult to say.

Marked physical improvement nearly always follows removal from conditions of poverty and filth to a well-ordered Asylum, irrespective of any treatment, and an improvement can be readily understood in the mental attitude of an impressionable race, such as form the bulk of our cases, when hope and interest are excited by a new treatment.

For example, by comparisons made between many cases which have shown signs of improvement since admission to the Asylum, it appears that the improvement made is the same in untreated cases as in those under treatment.

There is no doubt that leprosy is a disease of exceptional obstinacy, and also that its clinical and bacteriological conditions vary greatly from time to time quite independent of any treatment. The length of time during which these cases have been treated of itself introduces such an element of doubt that it is unfair to ascribe the small amount of improvement made to treatment, but rather to the general tendency of the disease towards a natural recovery.

Besides, were the nastin treatment possessed of any marked influence on the course of the disease, the larger number of cases which get progressively worse as compared with those showing improvement, in my opinion more than negatives any claim to a beneficial effect, especially when the decided improvement in the eight untreated cases in considered.

Careful clinical and bacteriological examinations made during and after practically two years' treatment, show, in a few cases only, certain changes towards improvement, but these changes are too slight to give any hope of recovery.

With regard to benzoyl chloride, its use as a nasal spray is of very great value: cases in which large numbers of leprosy bacilli were shown to be present in the nasal mucous membrane rapidly lost all traces of bacilli under the use of a $2\frac{1}{2}$ per cent. solution.

On broken-down nodules and ulcerating surfaces in which enormous numbers of bacilli were present it caused a rapid disintegration and destruction of bacilli.

Its subcutaneous injection seems to be of very doubtful utility.

In comparing the relative number of cases showing apparent improvement under benzoyl chloride with those treated with nastin, it must, however, be borne in mind that the patients treated with nastin contained a larger percentage of nodular cases than those treated with benzoyl chloride, the numbers being 45 per cent. for nastin and 35 per cent. for benzoyl chloride, a difference of 10 per cent.

Therefore, as the cases treated with benzoyl chloride show a 15 per cent. larger number of cases showing an apparent improvement over those treated with nastin, it would appear that the ratio of improvement should read as nearly equal, with a small margin of 5 per cent. in favour of the benzoyl chloride.

I beg to tender my thanks to the Medical Superintendent at the Leper Asylum for the assistance in carrying out these investigations.

Conclusions.

1. That nastin has apparently very little beneficial effect on cases of leprosy.
2. A solution of benzoyl chloride in oil shows a slightly higher percentage of improvement than nastin.
3. Anæsthetic cases of leprosy run a definite course, after which the disease seems to die out, leaving the patient no longer infective.
4. These cases recover sensation after a time in areas previously anæsthetic, and after self-amputation only scars remain.

   This is a natural process and takes place without any treatment whatever; it is not apparently influenced by either nastin or benzoyl chloride.
5. Nodular cases do not tend to improve naturally as above, except in very rare instances; nor do they appear to be affected appreciably by either nastin or benzoyl chloride.
6. The so-called destruction of bacilli is a natural process varying considerably, and does not appear to be influenced by nastin or benzoyl chloride.

7. The variations of the amount of destruction of bacilli observed are of limited value as an indication of the effects of treatment.
8. Benzoyl chloride in petroleum oil is extremely valuable as a nasal spray or a paint for ulcerating surfaces. It quickly renders the discharge free from the presence of bacilli.
9. Its regular use for this purpose is strongly recommended in Leper Asylums.

Notes on a Case of Pernicious Malaria complicated by Blackwater Fever.

By Captain O'DONOGHUE, R.A.M.C.

The patient, a private soldier, was sent to the Hospital on the morning of May 24th, 1909, at 8 A.M., with a note from the Adjutant to the effect that this man "fell out" on the parade ground as the regiment was starting out on a route march. On being questioned, patient stated that for the last few days he had suffered from severe pains in the back of his head and down along his spine, and that he had had no sleep the previous night as he was feeling so hot. His temperature was found to be 104° F., pulse 120. Two blood-slides were taken and sent to the laboratory for examination.

In the meantime patient was sent to bed and an ice-bag applied to his head, five grains of calomel were given by the mouth at 10 A.M. A telephone message from the laboratory reported that the blood contained an enormous number of ring parasites. At this time the patient was almost unconscious, being only roused with the greatest difficulty. His corneal reflexes had almost disappeared and his breathing was somewhat stertorous. Twenty-five grains of quinine bi-hydrochloride was given by intramuscular injection into both gluteal regions, and instructions were given to the Orderly-in-Charge that his temperature was to be taken every two hours, and if the temperature rose above 103° he was to be sponged with cold water. Before leaving the hospital at 1 P.M. I went to see the patient again. His temperature was then 103° F., his heart-beats were very irregular and the pulse rather rapid. I therefore gave him a hypodermic of strychnine. When I visited the hospital at 5 P.M. the patient's temperature was normal, having fallen from 103° to 98°. His mental condition was rather curious. He had a peculiar dazed expression of countenance combined with a certain amount of drowsiness. He would open his eyes and look round the ward, then at the Orderly and myself, then for a minute or two he would doze off only to waken again. It struck me that he was trying to collect his thoughts and also to remember where he was. On questioning him whether he knew where he was or did he know me he replied in the negative. Asked if he would like some milk he shook his head and then dozed off to sleep. His conjunctival reflexes were quite normal at this time. At 9 P.M. his condition was practically unchanged. His temperature was normal, but the drowsiness seemed to be more marked.

I gave him another intramuscular injection of 10 grains of quinine in the deltoid region.

May 25th, 1909.

This morning patient's temperature is normal. He complains of stiffness in the gluteal and deltoid regions. He states the pain in his head is much better but not quite gone yet. His bowels acted freely and he passed nearly 40 ozs. of urine, which was rather dark in colour. No albumen present. Patient was ordered 3 pints of milk and some Brand's essence of chicken. Two slides of blood were sent for examination to the laboratory with a request that a differential count might be done. The report was as follows:—

	per cent.
Parasites	Nil.
Lymphocytes	15·3
Large mononuclears	21
Polymorphonuclears	62
Eosinophiles	1·5

When I left the hospital at 1 P.M. patient was having some milk. He stated that he was feeling very well except that his head had been paining him more for the last few hours.

At 3.30 P.M. I got an urgent message from the Orderly to say that the patient was dying. When I arrived at the hospital about 20 minutes later the patient was absolutely comatose. His corneal reflexes were absolutely insensitive and nothing would rouse him. His rectal temperature was 106°·5 F. Time being of the greatest importance now, I thought the best and quickest way to give the quinine was by rectal injection, and as the patient had a good clearing out that morning no time was lost in giving a preliminary soap and water enema. A long tube was passed up the rectum and 40 grs. of the bi-hydrochloride was thrown up the rectum. At the same time an ice-bag was applied to the head and a lump of ice rubbed over the patient's body. That night at about 9 P.M. the temperature fell from 105° to below normal. At the same time he had a most profuse perspiration all over his body and his bed-clothes were simply soaked. He recovered consciousness at the same time and asked for a drink. When I saw him at midnight he was sleeping quite normally.

On visiting the hospital next morning, the Orderly informed me that the patient was all right but that he thought he had Blackwater Fever. On looking at the urine which he had just passed it was of an intense black colour, like porter, and had the same oily appearance as in a previous case of mine. On boiling it, it became practically solid. The patient seemed perfectly all right, except that his sclerotics were very yellow. He states that he had never had Blackwater Fever. On looking up the Admission and Discharge Books I find that he has had 15 admissions for malaria in the past 18 months, and on looking up his temperature charts I find that the type of fever was very similar, usually lasting about three days.

Patient was placed on the Hearsey treatment, viz. as much barley water as he could drink, and the following mixture to be given every hour:—

	grs.
Soda bicarb.	5
Hydrarg. perchlor.	1/60th
Water	2 ozs.

At 5 P.M. this afternoon patient had passed about 40 ozs. of urine, which was intensely black in colour, sp. gr. 1024, acid in reaction. A report from the laboratory as to the condition of his blood showed that it had not altered from the first. No parasites.

May 26th.

Patient spent a good night and passed about 63 ozs. of urine. On comparing it with that passed yesterday it seems much lighter in colour. Treatment continued.

May 27th.

Urine almost clear. Patient says he feels quite all right again. Has no headache and sleeps very well.

May 28th.

Urine perfectly clear. Sp. gr. 1015. No albumen.

A CASE OF AMŒBIC DYSENTERY OCCURRING IN A MAN WHO HAD NEVER BEEN OUT OF SCOTLAND.

[Major D. G. MARSHALL, I.M.S.—Edinburgh Medical Journal, March 1912.]

THE author points out that until recently it was believed that Great Britain, Scandinavia, Spain, and Portugal were the only countries in Europe from which cases of Amœbic Dysentery had not been reported as having been diagnosed during life, although a similar case, complicated by abscess of the liver, in a man who had never been out of England, occurred a few years ago in Birmingham and was reported by Saundby and Miller. In that case, unfortunately, the condition was not recognised during life, its true nature only being revealed at the post-mortem examination.

Major Marshall now records a case of the disease occurring in an agricultural labourer in Scotland, who had not been out of the country.

The patient was a ploughman, aged 28, residing near Dunbar, and was admitted to the wards of the Royal Infirmary, Edinburgh, in November 1911, suffering from chronic diarrhœa.

About seven weeks before admission he began to suffer from diarrhœa, the stools being of a very watery character and numbering three or four a day. The diarrhœa commenced suddenly one morning, the patient having been perfectly well the previous day. The stools for the first week did not contain blood, and defæcation was unaccompanied by pain during the first fortnight. Later the stools became "slimy" and contained whitish specks like pieces of mutton fat. As the disease progressed the stools became more numerous; they were slimy as before, and contained blood and whitish masses, evidently portions of mucous membrane. The patient stated that about a fortnight before admission he was free from diarrhœa for two days and did not pass any blood during that time.

On examination, though somewhat anæmic, the patient was found to be a well-developed muscular man. Nothing abnormal was found, except in the digestive system. He complained of continual uneasiness at the lower part of the abdomen, with severe griping and bearing-down pains before and directly after defæcation. The stools were watery and of a reddish colour owing to an admixture with blood, and containing white specks and shreds of mucous membrane with numerous amœbæ. The tongue was moist and clean. Abdomen: nothing abnormal on inspection; on palpation, pain was elicited on pressure over the region of the colon. Nothing abnormal to be found on rectal examination. The liver was not enlarged and there was no jaundice.

Nothing abnormal was to be seen in the blood-count, and the temperature was only above normal on one day during his stay in the hospital. Examination of the stools showed the presence of numerous amœbæ. The patient was treated with large doses of ipecacuanha, and by the end of December 1911 improved so much that he was allowed up. He was at that time passing one semi-solid stool a day. In a few days, however, his condition became as bad as before. Stools numbering six or seven a day and containing large quantities of blood were passed. He was again placed under ipecacuanha, and rectal injections of quinine were also administered.

Under this treatment the stools rapidly diminished in number and became free from blood, and by February 10th the patient was convalescent, passing only one stool a day and rapidly regaining his strength.

The author was unable to trace the source of infection with certainty.

The paper concludes with some valuable hints as to the method of examination of stools and the best way in which to stain the parasites. A short discussion on the various treatments for Dysentery which have been recommended from time to time is also added. H. B. N.

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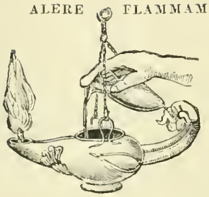

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THE LATE

ANDREW DUNCAN, M.D., F.R.C.P., Lt.-Col.(retd.)I.M.S.

THE Students of the London School of Tropical Medicine will have learned from the weekly medical papers, with profound regret, of the death of their old teacher, Dr. ANDREW DUNCAN. It is hardly necessary to amplify the various obituary notices that have already appeared; but we cannot allow this number of the School Journal to go to press without an expression, on behalf of the Staff of the School, of their sense of loss by the death of so highly esteemed a colleague.

Dr. Duncan, though perhaps not a brilliant lecturer, was a sound teacher, and one who spared no pains to keep his teaching abreast of the times. He excelled as a writer. His work 'The Prevention of Disease in Tropical and Sub-tropical Campaigns' was not only in advance of the times, but it is a model of what a medical work should be, with a vigorous style, lucidity, and arrangement. How it came about that one who entered the Indian Medical Service with so brilliant a record as Dr. Duncan could show, or one who did such brilliant work while in the Service, failed to attain the very highest place in that Service is difficult to understand. Possibly his independence of character and the uncompromising way in which he stuck to what he believed to be the truth may be the explanation. In those days official convenience and so-called policy counted for more than the truth. Though derided and slighted at the time they were propounded, Duncan had the satisfaction before his death, of seeing many of his ideas accepted and acted on at last.

Duncan was a teacher in the Tropical School from its commencement, and also a physician on the Hospital Staff. He took a great interest in the work of the School, and was most assiduous in the discharge of his hospital duties.

By none will his death be more deplored than by his colleagues, with whom he was always on the best and kindliest of terms. P. M.

I.—ENTOMOLOGICAL NOTES.

Description of a new Species of *Anopheles* from the Malay Peninsula.

By A. ALCOCK.

The School has received recently from Dr. A. T. Stanton two specimens of a remarkable species of *Anopheles*, which were bred by Dr. A. R. Wellington from larvæ found in a reservoir of drinking-water in the Larut Hills, in the Malay Peninsula. The reservoir lies at an elevation of 4000 feet and quite close to jungle.

Both specimens are female. One of them, besides being broken, is very much rubbed and mouldy; the colouring of its scutum is the only remaining character that surely proclaims its specific identity with its fellow. The other is perfect, except for a little mould and the want of one hind leg.

As a rule, it is improper to describe a species from a single specimen, unless it possesses some outstanding distinctive feature. As this specimen possesses, both metaphorically and literally, such a feature, and as, moreover, it bridges the stream of ink that separates the so-called genus *Myzorhynchus* from the so-called genus *Lophoscelomyia*, I venture to draw attention to it as a new species, under the name *Anopheles* (*Myzorhynchus*) *wellingtonianus*.

The type, in accordance with the custom of the School, will be deposited in the National Collection at Cromwell Road.

Anopheles (Myzorhynchus) wellingtonianus, sp. nov., ♀.

Head clad with the usual prominent dart-shaped scales, most of them black, but some, in the middle line anteriorly, white. Palpi and proboscis black, the palpi being shorter than the proboscis and shaggily scaled.

The greater part of the scutum forms an elegant suboval field of a warm lavender-grey colour (peach-colour in life), which is longitudinally divided by a fine brown streak, and is very sharply defined from the side-borders of the scutum, which are rich purplish black. The covering of the scutum consists of sparse hairs.

In the legs all the coxæ are pallid and the other joints are purplish brown to black, the extreme tips of the femora and tibiæ only being pale. The femora of the hind legs are distinguished by the presence of a very broad smooth band of small and closely adherent white scales, which runs into a prominent subterminal brush, or ruff, of long black scales. This ornamentation of the hind femora is much like that of *Anopheles asiaticus* ("*Lophoscelomyia asiatica*"), except that in the latter species the white scales are salient (not tightly adherent) and distal (not proximal).

Wings thickly clothed as to the veins with short elliptical scales of some breadth; most of the scales are black, but there are, in addition to some spots in the fringe, ten spots or patches of yellow or yellowish scales, situated as follows:—(1) on the anterior costa just beyond the middle, small: (2) near the tip of the wing, large; (3) in the distal half of the posterior branch of the 2nd vein, large; (4) near the origin of the 3rd vein; (5) at the proximal end of the anterior branch of the 4th vein, whitish; (6) at the tip of the posterior branch of the 4th vein, whitish and very small; (7) at the fork of the 5th vein; (8) near the proximal end of the anterior branch of the 5th vein, whitish; (9) near the distal end of the same branch of the same vein, whitish; (10) at the tip of the 6th vein, whitish and very small. There are three fairly distinct yellowish breaks in the posterior fringe of the wing, situated respectively at the tips of the two branches of the 5th vein and of the posterior branch of the 4th.

Halteres with pale stem and dark knob.

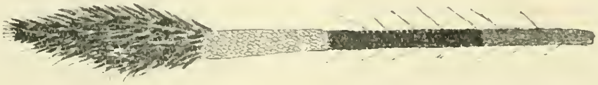

Fig. 1.—Femur of hind leg of *Anopheles* (*Myzorhynchus*) *wellingtonianus.*

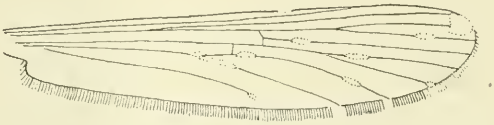

Fig. 2.—Diagram of wing of *Anopheles* (*Myzorhynchus*) *wellingtonianus*, to show the position of the yellow (some of them whitish) spots.

Abdomen purplish brown, rather smudged or rusty in places; fairly well beset with hairs, which in the middle of some of the terga are shorter than elsewhere, but are still very much too long to be called scales. In the middle line of the 7th abdominal sternum is a single outstanding scale, which may perhaps be the remains of a tuft.

Anopheles wellingtonianus has many points of resemblance both to "*Lophoscelomyia*" *asiatica* and to *Anopheles* (*Myzorhynchus*) *barbirostris*. It certainly supports the opinion that whatever view be taken of the limits of the genus *Anopheles* all three species are congeneric.

THE ANOPHELES MOSQUITOES OF MALAYA AND THEIR LARVÆ, WITH SOME NOTES ON MALARIA-CARRYING SPECIES.

By A. T. STANTON, M.D.,

BACTERIOLOGIST, INSTITUTE FOR MEDICAL RESEARCH, FEDERATED MALAY STATES.

SINCE the publication of the monograph by Drs. G. F. Leicester and C. W. Daniels on "The Culicidae of Malaya" in 1908, several new observations have been made which appear to be of sufficient value to justify a revision of the group of Malayan Anopheles. An examination of specimens from different countries in the Oriental region and of the types preserved in the British Museum of Natural History has shown that much confusion still exists in mosquito nomenclature. In certain cases the same mosquito is known under different names—for example, *fuliginosus* of India is identical with *nivipes* of Malaya; while in other cases different mosquitoes are known under the same name—for example, *willmori* of Malaya (Leicester) is different from *willmori* of India (James and Liston). One regrettable result of this confusion is that the knowledge gained of malaria-carrying species in one country is rendered valueless in its application to the circumstances of the other.

The present paper embodies the results of a study of a large series of Malayan Anopheles and comparison of these with specimens of the Indian species and with the type specimens at the British Museum. The individual species are not described, but a table is appended showing how those may be recognised which are considered to be of undoubted validity.

In order to avoid the vexed question of the generic classification of Anophelines, all the species here dealt with are referred to the genus *Anopheles*. In a department of zoology where so much uncertainty still exists as to the characters of species, it seems little profitable to complicate matters further by attempting at present to define the limits of genera or subgenera.

In those cases in which the mature forms of the larvæ are known, a brief description is given of the characters which are of value in the differentiation of species. These characters are in particular (see figs. 1 & 2, p. 5) the form and arrangement of (1) the four anterior clypeal hairs, here called the inner and outer pairs of anterior clypeal hairs, and (2) the two posterior clypeal hairs, both of which groups are situated on the front of the head, and (3) the palmate hairs, situated laterally on the thorax in some species and on a varying number of the abdominal segments.

In the course of the present study it was discovered that, contrary to what had hitherto been believed, the form and arrangement of the clypeal hairs of Anopheles larvæ varied at different stages of the larva's life. The observations made in connection with this matter will be considered more in detail in another place, but it may here be mentioned that when they leave the eggs Anopheles larvæ of whatever species are very much alike (at any rate in those features which are commonly supposed to exhibit constant specific differences), and that it is only in the later stages that their distinctive characters are developed. These facts explain the anomalous results obtained by previous workers in this field of study.

ANOPHELES AITKENI, James, 1903.

Anopheles aitkeni, James in Theobald, 1903.
Stethomyia fragilis, Theobald, 1903.
Anopheles treacherii, Leicester, 1908.
Neostethopheles aitkeni (James & Liston, 1911).

This is the only unspotted winged Anopheles so far recorded from the Malay Peninsula. It is remarkable for its frailness and for its culex-like attitude when at rest.

This species has been suspected to be a transmitting agent of malaria, but there is no evidence in support of this suspicion.

Larva.—The larva is short, and the unusually broad thorax gives it a stumpy appearance. The inner anterior clypeal hair is frayed in its basal two-thirds and simple at the end: the outer anterior clypeal hair is simple; the posterior clypeal hair is branched. Palmate hairs are present on the thorax and on the first to seventh abdominal segments.

ANOPHELES UMBROSUS, Theobald, 1903.

Myzorhynchus umbrosus, Theobald, 1903.

This species usually shows only one white spot on the wing costa, but in many specimens there are two spots. The latter variety is stated to be a malaria carrier. Five trials by the writer failed to show any development of the parasites of subtertian malaria in this species.

Larva.—The inner anterior clypeal hair is simple; the outer anterior clypeal hair is branched, four or five divisions springing from a single stem; the posterior clypeal hair is branched. The antenna bears a long branched hair on its inner side. No leaf-like palmate hairs are present on either thorax or abdomen; this remarkable character was not noted in the mature larva of any other species of Anopheles.

ANOPHELES ASIATICUS, Leicester, 1904.

Lophoscelomyia asiatica, Leicester, 1904.

This species is recorded by Dr. Leicester as "exclusively a bamboo breeder." The larva has not yet been described. A specimen of Anopheles recently received by the writer from Dr. A. R. Wellington, of Perak, would appear to indicate that this species has affinities with the *umbrosus* group of *Anopheles*.

ANOPHELES ALBIROSTRIS, Theobald, 1903.

Myzomyia albirostris, Theobald, 1903.

This is a common species in certain parts of the Peninsula; it resembles the Indian species *listoni* in appearance, habitat, and in the readiness with which it may be infected with malaria parasites. It has been found infected in nature (zygotes), and the parasites of subtertian malaria readily undergo development in it. It is believed to be an important agent in the spread of malaria in the Peninsula.

Larva.—A short, stumpy larva resembling that of *A. fragilis*. Both outer and inner anterior clypeal hairs are pinnate; the posterior clypeal hair is branched. Palmate hairs are present on the thorax and on the first to seventh abdominal segments; on the thorax and first abdominal segment these are lanceolate, on the other abdominal segments they are broad and jagged at the base of the terminal filament, which is long and sharp-pointed (fig. 3).

NOTE.—With reference to the species *aurirostris*, Watson, 1910, which is said to differ from *albirostris*, I have not taken any specimens which answer to the description.

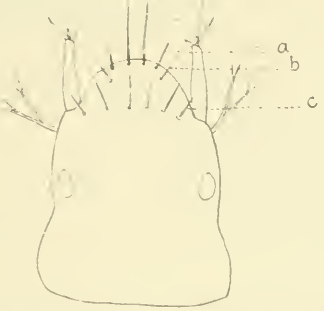

Fig. 1.—Head of new-hatched larva of *Anopheles albirostris* to show *a*, anterior clypeal hairs; *b*, posterior clypeal hairs; *c*, frontal hairs.

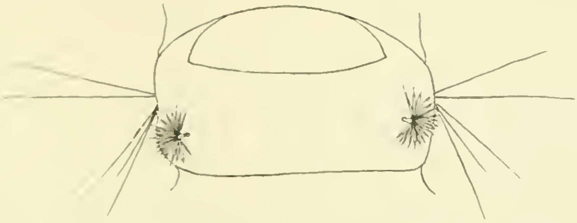

Fig. 2.—Fifth abdominal segment of full-grown larva of *Anopheles albirostris* to show the cockades or "palmate hairs."

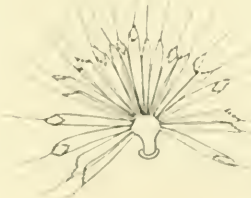

Fig. 3.—One of the cockades of the same, further enlarged.

ANOPHELES ROSSI, Giles, 1899.

Myzomyia rossi (Theobald, 1903).
Pseudomyzomyia rossi (Theobald, 1907).
Nyssomyzomyia rossi (James & Liston, 1911).

This is a very common species everywhere throughout the Peninsula. A large series of dissections and infection experiments failed to show any development of malaria parasites in it.

Larva.—Both anterior clypeal hairs are simple, the inner being long and stout, and the outer very short; the short posterior clypeal hair is simple and is situated internally to the line of the inner anterior clypeal hair. The thorax does not carry palmate hairs—these are present on the first to seventh abdominal segments, the terminal filament being unusually long and sharp-pointed.

NOTE.—With reference to the species *Anopheles indefinatus*, Ludlow, to the description of which a number of Malayan specimens correspond, it should, perhaps, be considered as a variety of *rossi*, rather than as a distinct species.

ANOPHELES LUDLOWI, Theobald, 1903.

Myzomyia ludlowii (Theobald, 1903).
Nyssomyzomyia ludlowi (James & Liston, 1911).

This species is also a near ally of *rossi*. In the Malay Peninsula it has been taken only in the coast towns.

New interest has been aroused in this species owing to Major Christopher's announcement that he has found it to be a carrier of malaria in the Andamans.

Larva.—No constant difference has so far been noted between the mature larva of *ludlowi* and that of *rossi*.

ANOPHELES TESSELLATUS, Theobald, 1901.

Anopheles tessellatum (Theobald, 1901).
Anopheles punctulatus, Theobald nec Dönitz, 1901.
Anopheles tessellatus (Theobald, 1901).
Myzomyia tessellatum (Theobald, 1903).
Nyssomyzomyia punctulata (James & Liston, 1911).

In the first volume of his monograph, Mr. Theobald describes an *Anopheles* from Perak, Federated Malay States, for which he proposed the name *tessellatum*. Before publication, having seen the description of *punctulatus*, Dönitz, he considered the two to be synonymous and used his description as that of *A. punctulatus*, Dönitz. It has now been determined that the two species are distinct, one marked difference being in the palpal banding. Therefore Mr. Theobald's description of what he called *A. punctulatus*, Dönitz, stands good for his own *tessellatus*, which now ranks as a good species.

This is a comparatively uncommon mosquito in the Malay Peninsula and its larva has not yet been identified, nor has any investigation been made of its malaria-carrying abilities.

ANOPHELES LEUCOSPHYRUS, Dönitz, 1901.

Myzomyia ? elegans, James in Theobald, 1903.
Pyretophorus elegans (Theobald, 1907).
Neomyzomyia elegans (Theobald, 1910).
Myzomyia "leucophyrus," Leicester, 1908.

Examination of Malayan specimens of Leicester's "*leucophyrus*," Dönitz, shows them to be identical with Indian *elegans*, James, and with *leucosphyrus*, Dönitz.

This species has been met with very infrequently.

Larva.—Both anterior clypeal hairs are simple: the posterior clypeal hair is simple. The thorax does not carry palmate hairs, but these are present on the first to seventh abdominal segments; the leaflets are broad and the terminal filament short and blunt.

ANOPHELES MACULATUS, Theobald, 1901.

Anopheles maculata, Theobald, 1901.
Nyssorhynchus maculatus, Theobald, 1903.
Nyssorhynchus willmori, Leicester nec James, 1908.
Nyssorhynchus pseudowillmori, Theobald, 1910.

The nomenclature of this and closely allied species has given rise to more difficulties than any others of Oriental Anopheles. An examination of the types of *Anopheles maculata*, Theobald, has revealed a reason for the confusion that has existed in the minds of Eastern workers as to the characters of this species. The types are not male and female of the same species, but represent two distinct species, the male being of the species known to Eastern workers as *maculatus* and the female of the species known to them as *karwari*.

Dr. Leicester in his monograph, page 44, referring to his *Nyssorhynchus willmori*, says: "This mosquito evidently bears a strong resemblance to *Nyssorhynchus maculatus*, specimens of which have been taken at Taiping. It differs, however, in the banding of the palpi. A drawing of the palpi of *maculatus* is given in Theobald's monograph and shows three white bands at the apex instead of two equal bands present in *willmori*." Dr. Leicester's reference to the drawing is to that on page 101, vol. iii. of Mr. Theobald's monograph, which is in reality a representation of the female palp of the species known to him as *karwari*. In consequence of these difficulties Dr. Leicester makes the quite excusable error of assigning this common Malayan Anopheles to the species *willmori*, an error in which he has been followed by later workers, to the production of endless confusion.

The zoological puzzle represented by these facts it is proposed to solve by retaining the name *maculatus* for the species which is now well known under that name to students of this group in the Orient, and to rename the species which has hitherto been known as *karwari*. It is admitted that this course is open to criticism, on the ground of nonconformity with the strict rules of zoological nomenclature, but other considerations move me to regard it as the most satisfactory.

This species has been found infected in nature both by Dr. M. Watson and by the writer. I have also been able to infect it under experimental conditions with the parasites of subtertian malaria.

Larva.—The inner and outer anterior clypeal hairs are pinnate, the posterior clypeal hair is simple. Palmate hairs are absent from the thorax and first abdominal segment, rudimentary on the second abdominal segment, and fully developed on the third to seventh abdominal segments: the leaflets are broad and notched at the base of the terminal filament, which is short and blunt.

ANOPHELES NIGRANS, nom. nov.

Nyssorhynchus karwari, James in Theobald, 1903.

The name of this species is altered for reasons stated under the account of *A. maculatus*.

It is a common Malayan species, the larvæ of which are frequently found in association with those of *maculatus*. It is stated to be a malaria carrier, but as yet no experimental or other evidence has been recorded in support of this suggestion.

Larva.—The description of the mature larva of *maculatus* applies also to this species. Some minor differences were observed, but it was not possible to satisfy oneself that these were constant.

ANOPHELES FULIGINOSUS, Giles, 1900.

Anopheles leucopus, Dönitz, 1901.
Anopheles jamesii, Liston, 1901.
Nyssorhynchus nivipes, Theobald, 1907.

This species has been found infected in nature both in India by J. R. Adie and in Malaya by the writer, who has also observed the development in it of the parasites of subtertian malaria (zygotes).

Larva.—The inner anterior clypeal hair is much frayed in its terminal three-quarters, the outer anterior clypeal hair is thickly branched ; the posterior clypeal hair is branched. Palmate hairs are present on the thorax and on the first to seventh abdominal segments ; on the thorax and first abdominal segments these are lanceolate, on the second to the seventh segments they are broad and jagged at the base of the terminal filament, which is long and sharp-pointed.

ANOPHELES SINENSIS, Wiedemann, 1828.

Anopheles jesoensis, Tsuzuki, 1902.
Myzorhynchus peditæniatus, Leicester, 1908.

Tsuzuki states that in Japan this species is the carrier of benign tertian malaria. In the Federated Malay States I have seen malaria zygotes in it on two occasions, but was unable to infect it under experimental conditions with the parasites of subtertian malaria.

Larva.—The inner anterior clypeal hair is long and simple, the outer anterior clypeal hair is brush-like. The antenna bears a long branched hair on its inner side. Palmate hairs are present on the thorax and on the first to seventh abdominal segments.

NOTE.—With reference to the species *Myzorhynchus separatus,* Leicester, 1908, I have been unable to satisfy myself by an examination of the scanty material now available that it is distinct from *A. sinensis.* Leicester's *peditæniatus* is considered to be a synonym of *sinensis,* as are also *annularis* Van der Wulp, *vanus,* Walker, and *minutus,* Theobald, which have also been recorded from the Malay Peninsula.

ANOPHELES BARBIROSTRIS, Van der Wulp, 1884.

Myzorhynchus barbirostris (Theobald, 1903).

A feature of the Malayan specimens of *barbirostris,* which is not usually noted in descriptions of this species, is the presence on the ventral aspect of the second to seventh abdominal segments of a few white scales. On the second segment these scales are situated on either side of the mid-line and on the other segments in the mid-line. Like the ventral tuft of scales on the eighth segment, these scales are absent in males.

In eight trials it was not possible to infect this species with the parasites of subtertian malaria.

Larva.—In all its constant characters the larva resembles that of *sinensis*.

Anopheles albotæniatus, Theobald, 1903.

Myzorhynchus albotæniatus, Theobald, 1903.

This species was described by Mr. Theobald from material sent him by Dr. M. J. Wright, of Perak, Federated Malay States. It bears a general resemblance to *umbrosus*, but the broad banding of the hind tarsi is a distinctive feature. The name is retained for the present and pending an examination of further material.

Anopheles kochi, Dönitz, 1901.

Cellia kochii (Theobald, 1903).
Christophersia halli, James, 1910.

Though this species is suspected on epidemiological grounds to be a carrier of malaria, no evidence was obtained to support this view in the few experiments undertaken.

Larva.—The inner anterior clypeal hair is long and slightly frayed at the end, the outer anterior clypeal hair is short and simple; the posterior clypeal hair is simple. Palmate hairs are present on the thorax and on the first to seventh abdominal segments—on the thorax, first and second abdominal segments these are lanceolate, on the third to seventh segments they are broad and jagged at the base of the filament, which is short and blunt.

Table showing Principal Distinctive Characters of Female Malayan Anopheles.

I. With unspotted wings.

aitkeni	A small brown fragile species. At rest has a culex-like attitude.

II. With spotted wings.

A. With unbanded palpi.

umbrosus	Wings usually with only one costal spot and one small apical fringe spot. Palpi lightly scaled. Abdomen without ventral tuft or other scales. Hind tarsi narrowly banded.
albotæniatus	Wings with two costal spots and black fringe. Abdomen without scales. Hind tarsi broadly banded.
barbirostris	Wings with two costal spots, one pale apical fringe spot and another opposite lower branch of fifth vein. Palpi heavily scaled. Abdomen with ventral tuft of scales on eighth segment and scattered white scales on second to seventh segments. Hind tarsi narrowly banded.
asiaticus	A species with a distinctive ruff of black and white scales at the apices of the hind femora.

B. Palpi with five white bands.

kochi	A golden-brown species with six prominent tufts of black scales projecting downwards from ventral surface of abdomen.

C. Palpi with four white bands.

sinensis	Four narrow bands. Ventral tuft of scales on eighth abdominal segment.
tessellatus	Three broad bands and one narrow band. Speckled legs.
leucosphyrus	One broad apical band and three narrow bands. Speckled legs. Broad white band on hind legs involving lower end of tibia and upper part of first tarsal segment.
nigrans	Two broad bands and two narrow bands. Legs not speckled. Three broad bands on hind tarsi.

D. Palpi with three white bands.

maculatus	Two broad bands and one narrow band. Legs speckled. Three broad bands on hind tarsi.
albirostris	Two broad bands and one narrow band. Legs not speckled. Hind legs narrowly banded.
fuliginosus	One broad band and two narrow bands. $3\frac{1}{3}$ segments of hind tarsi white.
rossi	One broad band and two narrow bands. Legs not speckled. Hind tarsi narrowly banded.
ludlowi	One broad band and two narrow bands. Legs speckled. Hind tarsi narrowly banded.

Table showing Principal Distinctive Characters of *Mature* Larvæ of Malayan Anopheles.

A. With none of the anterior clypeal hairs simple.

albirostris	Anterior clypeal hairs pinnate. Posterior clypeal hair short, branched. Palmate hairs present on thorax and first to seventh abdominal segments.
maculatus	Anterior clypeal hairs pinnate. Posterior clypeal hair long, sometimes forked. Palmate hairs not present on thorax or first abdominal segment.
nigrans	Not distinguishable from *maculatus*.
fuliginosus	Inner anterior clypeal hair frayed, outer thickly branched. Posterior clypeal hair branched. Palmate hairs present on thorax and first to seventh abdominal segments.

B. With inner anterior clypeal hairs only frayed.

aitkeni	Inner anterior clypeal hair much frayed in its basal two-thirds. Posterior clypeal hair branched. Palmate hairs present on thorax and first to seventh abdominal segments.
kochi	Inner anterior clypeal hair slightly frayed at the end. Posterior clypeal hair simple.

C. With outer anterior clypeal hairs only branched.

umbrosus	Outer anterior clypeal hair slightly branched. No leaf-like palmate hairs on thorax or any abdominal segment.
sinensis	Outer anterior clypeal hair thickly branched. Leaf-like palmate hairs on thorax and first to seventh abdominal segments.
barbirostris	Not distinguishable from *sinensis*.

D. With all the anterior clypeal hairs simple.

rossi	Short posterior clypeal hairs between the inner anterior clypeal hairs. Terminal filament of palmate hairs long and sharp-pointed.
ludlowi..........	Not distinguishable from *rossi*.
leucosphyrus	Posterior clypeal hair behind inner anterior clypeal hair. Terminal filament of palmate hairs short and blunt.

In conclusion, I desire to acknowledge my indebtedness to Lt.-Col. A. Alcock, C.I.E., F.R.S., for much valuable advice and assistance in the preparation of these notes, and to the authorities of the British Museum and of the Central Malaria Bureau of India for the opportunity of examining specimens.

II.—DEPARTMENT OF PROTOZOOLOGY.

Experiments on the Behaviour of Leishmania and Allied Flagellates in Bugs and Fleas, with some Remarks on Previous Work.

By C. M. WENYON, M.B., B.S., B.Sc.,

Protozoologist to the London School of Tropical Medicine.

It was first shown by Patton that the parasite of Indian Kala Azar would develop into flagellates in the gut of *Cimex rotundatus*. More recently Patton has repeated these experiments not only with *Cimex rotundatus* but also with *Cimex lectularius*. In the latter series of experiments he found that the flagellate forms first produced give rise ultimately to small post-flagellate forms which have no flagellum. It is these post-flagellate forms which Patton thinks enter the body when the infected bug bites. In this manner, probably by a regurgitation from the stomach of the bug during its feed, the post-flagellate forms give rise to Kala Azar. The direct transmission of Kala Azar to animals by the bug has not been done.

In 1910 I discovered that the bed-bug of Bagdad (*Cimex rotundatus* ?) would take up leishmania from Oriental Sore and that in it the parasites developed into flagellate forms. This observation was later confirmed by Patton, who was working with Oriental Sore in Cambay in India. Patton came to the conclusion that the bug was also the transmitter of Oriental Sore. In my report on Oriental Sore I contested the view that the bed-bug was the transmitter of Oriental Sore and Kala Azar, and explained the development which took place in the bug as comparable to that which the leishmania would undergo when introduced into blood agar media in the test-tube.

Will Leishmania tropica *develop in* Stegomyia fasciata ?

In his last report on the development of the parasite of Indian Kala Azar in the bug Patton points out that my view cannot be the right one, since he has shown that various other blood-sucking arthropoda which take up a large quantity of blood with the leishmania do not permit of these parasites developing to the flagellate stage. The arthropoda mentioned are *Culex fatigans*, *Neocellia stephensi*, *Stegomyia sugens*, *Ornithodorus savignyi*, *Pediculus capitis*, and *Pediculus vestimenti* It is also pointed out that the parasite of Cambay boil will not develop in *Stegomyia*. In Bagdad I conducted experiments with *Stegomyia fasciata*, and found that about 10 per cent. of such *Stegomyia* which had fed upon the sore were found to have taken up leishmania when dissected immediately afterwards; and in six of these mosquitoes out of eighty which had fed upon the sore and were dissected at intervals after feeding, round and flagellate forms were seen which I concluded were most probably developmental forms of leishmania; while of over seventy *Stegomyia* which had fed upon a healthy person and were dissected as controls, none were found to harbour any flagellates. It will be seen that the percentage of mosquitoes which had taken up parasites is about equal to the percentage of those found infected when dissected some time after feeding. This in itself is great presumptive evidence in favour of the flagellates seen in the *Stegomyia* being in reality developmental forms of leishmania. Now Patton doubts very much whether the flagellates seen by me were flagellate forms of leishmania, chiefly because he obtained no evidence of such a development with the *Stegomyia* in Cambay. It is stated that a large number were fed on the case, some on the margin of the sore and some on distant parts. We are not told how many mosquitoes were used, nor if by immediate dissection after feeding any of the *Stegomyia* were proved to have taken up parasites. Unless it is known that the *Stegomyia* can take up parasites when feeding in the way adopted for experiment, it is useless to state that the leishmania will not develop into flagellates. Patton states that he was unable to find any parasites changed or unchanged in his mosquitoes, so that he has no evidence whatever that the *Stegomyia* had taken up parasites when feeding. His method of feeding the *Stegomyia*, either because the case was not a good one or else the mosquitoes fed too far away from the lesion, was evidently not one which would enable the mosquitoes to take up leishmania. In my experiments the mosquitoes fed on top of a non-ulcerating sore, and, as I have stated, at least 10 per cent. of these took up parasites directly from the sore; and when one finds that about 10 per cent. of *Stegomyia* fed upon the sore and dissected after varying intervals show forms which resemble developmental forms of leishmania, the most reasonable conclusion is that they are really such, especially when an equal number of *Stegomyia* fed upon a healthy person gave negative results when dissected after similar intervals. The fact that the larva of *Stegomyia* may contain a flagellate does not really affect the issue, since it was shown that this did not appear in any of the control mosquitoes dissected. Patton does not tell us if the larva of the Cambay *Stegomyia* may harbour such a flagellate.

The Gut of the Bed-bug as a Culture-tube for Flagellates.

Now Patton's failure to obtain a development of the *Leishmania donovani* in the arthropoda mentioned above and also in *Conorrhinus rubrofasciatus* is in itself no proof that the leishmania develop in the bed-bug in any other way than they do in the blood agar medium. Though leishmania will not flagellate in these arthropoda, it is still possible that they develop in the bug as they do in the culture-tube. It may simply mean that these arthropoda have some substance in the gut which is injurious to the leishmania (this must be so), while the gut-contents of the bug has no such substance. As a matter of fact, there is considerable evidence of the inocuous nature of the gut contents of the bug.

1. I have shown that the spirochætes of relapsing fever when taken up by the bug will remain active for as much as seven days and appear normal in stained films after three weeks in the bug's gut *.

2. It has recently been shown by Brumpt that *Schizotrypanum cruzi*,

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* See Graham V. Smith, "On some Cases of Relapsing Fever in Egypt and the Question of Carriage by Domestic Vermin," p. 36. John Bale, Sons, & Danielsson, Ltd., London.

whose natural host is a species of *Conorrhinus*, will undergo a development in the bed-bug, and that the forms which are found both in the mid and hind gut are infective for ten days when injected into animals. Not only does *Schizotrypanum cruzi* develop in *Cimex lectularius* but also in *Cimex boueti* and *Ornithodorus moubata*, which are all unable to transmit the infection as does the true host of the *Schizotrypanum*.

3. It was shown by Swellingrebel and Strickland that *Trypanosoma lewisi* will undergo a development in *Cimex lectularius*, and that as late as nine days crithidial forms occur in the mid gut. I have repeated this observation and have found that on the eleventh day trypanosome, crithidial, herpetomonas, and leishmania forms occur. Some of the typanosomes bear a striking resemblance to the small infective forms which appear in the hind gut of infected fleas. It is hardly necessary to say that the bug is not the host of *T. lewisi*.

4. I have fed bugs on rats infected with *Trypanosoma rhodesiense*, and after seven days not only can trypanosome and crithidial forms be found, but also many rounded parasites. The trypanosomes, however, disappear very rapidly from the intestines of fleas.

It is thus quite clear that the gut of the bug when it contains blood acts as a culture-tube in which various organisms can live and develop. This must depend to a great extent on the absence of any harmful material which might destroy them. It is hardly to be wondered at that such an easily cultured organism as the leishmania should also undergo a development in the bug's gut. Since we know that other flagellates will develop here, it would be a remarkable fact if the leishmania did not so develop. As a matter of fact, the persistence of the flagellates in the gut of the bug appears to be in proportion to the ease with which they are to be cultured in the blood agar medium. Hence the fact that the leishmania of Kala Azar and Oriental Sore become flagellates in the bug's gut is in itself no proof whatever that these insects are the transmitters of these diseases. Patton has stated that in his opinion the parasite of Mediterranean Infantile Kala Azar will be found to flagellate in *Cimex lectularius*. I will extend this prophesy and include the leishmania of Canine Kala Azar and of Dermal Leishmaniasis of South America.

The Final Stage of Development of Leishmania in the Bed-bug.

In his last report on the parasite of Kala Azar, Patton lays great emphasis on the appearance in the bugs of what he calls the post-flagellate forms, which are rounded forms developed from the flagellate forms. In the life-history of a typical *Herpetomonas* of any insect such rounded forms are developed from the flagellates in the hind gut, and it is these which, protected by some kind of covering, pass out in the faeces, and are responsible for the transmission of the infection to a new host. The round forms of leishmania which appear in the bugs after the eighth day are compared by Patton to the post-flagellate forms of a *Herpetomonas*, and on that account he is convinced that he has traced the whole development of the leishmania from the forms ingested through the flagellate stage to the post-flagellate. In the bug, however, the post-flagellate is not formed in the hind gut as is usually the case with the true *Herpetomonas* of insects, but in the mid gut, and it is supposed that they gain entrance to a new human host by regurgitation when the bug bites. Whether the forms seen in the bug are true post-flagellate forms in the same sense as those that occur in insects must be an exceedingly difficult matter to decide. Patton, who has had much experience with the *Herpetomonas* of insects, says he can recognise them by their peculiar staining reaction. A staining reaction, especially by a Romanowsky stain and in such a variable medium as the gut contents of an insect, is hardly a reliable test. Further, it is known that in cultures of leishmania in blood agar there is first the production of a flagellate stage and ultimately the production of round forms without a flagellum. Row has insisted on the fact that these round forms are really post-flagellates ; but whether this is so or not it is evident that the ultimate production of round forms in the mid gut of the bug is a further illustration of the similarity of the behaviour of the leishmania in the culture-tube and in the bug's intestine. Patton has shown that if the bug during the stage in which flagellate forms of leishmania are present in its gut has a second feed of blood, the flagellate forms are destroyed, and this he attributes to the presence of complement in the blood. It is this complement which explains in his mind the absence of flagellate forms in the body of man, and he claims that the round post-flagellate forms must be produced before infection can take place. It has been frequently shown that artificial cultures of leishmania may produce both Oriental Sore and Kala Azar when injected into animals. It must then be some more resistant form in these cultures—a post-flagellate—which causes the infection, for, according to Patton, the flagellate forms are destroyed by the blood. It thus appears that the development of the leishmania in the bug is a picture—and, as a matter of fact, a somewhat restricted picture—of the development undergone by leishmania in the blood agar media. Just as in the test-tube when the culture is old and nourishment is exhausted round forms appear, so in the bug the so-called post-flagellates are produced when the blood taken up by the bug is reaching the completion of its digestion.

The Development of Leishmania in the Bug is a very restricted one.

Another point which to my mind is a very strong argument against the bed-bug being the true host of these organisms is the very restricted development undergone by them in the bug. Experiments with Oriental Sore are much more difficult to conduct than with Kala Azar, in which comparatively large numbers of parasites may be present in the peripheral blood. When a bug feeds upon the sore one has no criterion as to how many parasites have been taken up, but in the case of the peripheral blood of Kala Azar cases it would be possible by measuring the quantity of blood taken up by a bug to estimate fairly accurately the number of parasites it had ingested. When, for instance, as many as 250 parasites occur in a blood-film, it must be certain that a bug feeding on the case had taken up a considerable number of parasites. Now the point I wish to emphasise is this : namely, that the extent to which the bug becomes infected—or, in other words, the number of flagellates to be found in its gut after a few days—varies directly with the number of parasites ingested. In experiments on Oriental Sore, where the number of leishmania taken up must be very small, one never finds a large infection with flagellates ; while in Patton's last experiments, in which the bugs had fed upon blood from a heavily infected case of Kala Azar with numerous parasites in the peripheral blood, he obtained large numbers of flagellates. If the bug were the true host of the leishmania a complete infection of the gut would take place whether many or few parasites had been ingested: the only difference would be that the bug would take a longer time to show a large infection when the parasites were few. In the case of insects which are infecting themselves with herpetomonas by the ingestion of post-flagellate forms voided by another, it is surely not necessary for the insect to take up large numbers of these in order to become heavily infected. Presumably the ingestion of one such post-flagellate form would be sufficient to give rise ultimately to the heavily infected gut so commonly seen in insects harbouring herpetomonas. If the bug were the true host of leishmania it would be expected that the ingestion of only a few parasites would be sufficient to produce a large infection of the gut.

The Effect of a Second Feed of Blood on the Flagellates in Insects.

Patton has shown that a second feed of blood is fatal to the flagellate forms of the leishmania which may have developed in the bug. This, again, appears to me to be an argument against the bug hypothesis. I have recently kept under observation fleas infected with herpetomonas and others infected with *Trypanosoma lewisi*. One flea has been observed for two months, during which time it has had sixteen feeds of human blood. By an examination of the fæces voided during the feeding act I have found that it has constantly remained infected with herpetomonas, which were passed in the fæces in very large numbers at every feed, so that a constant active multiplication must have gone on. Two fleas infected with *Trypanosoma lewisi* were similarly fed on human blood during the course of one month. The fæces constantly showed the small infective trypanosomes characteristic of *T. lewisi*. Why, then, did not the constant feeding destroy the flagellates in the guts of these animals? The explanation must be that the flagellates in question were in their true host and were accustomed to human blood. This should be true of the leishmania in its true host, and the disappearance of the herpetomonas forms when the bug has a second feed simply shows that the flagellate is in an unnatural environment. Patton has shown that the natural herpetomonas of *Culex fatigans* disappears from this insect when it has a feed of blood. This is not remarkable if we remember that the herpetomonas is really a flagellate of the larva of the mosquito, which is not a blood-sucker, and remains in the adult only so long as the intestinal contents approximate in composition to those of the larva. There is one observation which might be produced as an argument against this contention. Miss Robertson has shown that batches of *Glossina* fed upon cases of human trypanosomiasis show a greater percentage of individuals infected if they have had only a single feed of blood. The explanation of this is at present not clear, for we know that an infected *Glossina* may infect a series of animals at intervals of several days.

Patton sees in this effect of a second feed of blood an explanation of the tendency of Kala Azar to remain localised in Madras and Oriental Sore in Cambay, and says: "This puzzling fact in connection with the disease can now be better explained, when it is known that though many bugs may become infected, only those which do not feed again till the parasite has passed back to its post-flagellate stage are infective; the number capable of transmitting the parasite must then necessarily be reduced." There is in this assumption a fallacy of fundamental importance. In every case where the intermediate host is well known, it is not this which is the prime cause in the spread of the disease outside any given locality. It is not, as a rule, the intermediate host which transports the infection from place to place, but the reservoir, which is frequently man himself, who passes from one locality where the disease exists to another where the necessary intermediate host already occurs. Trypanosomiasis spreads because infected individuals travel, and not because the *Glossina* spreads the infection ; Yellow Fever is similarly spread rather by the moving about of infected persons than by the transportation of the infected *Stegomyia* ; Plague is carried by the rats and Malaria by man ; and in the case of Kala Azar there is nothing to prevent the infected individual moving from Madras to another town where bugs are ready to feed and become infected. In man the infection is of long duration, while there is no evidence to show that bugs remain infective for any lengthy period, even if we assume they are so at any time. It must constantly happen that people suffering from Kala Azar and Oriental Sore move from Madras and Cambay respectively to other localities where bugs abound. Why, then, do not these cases become the starting-point in the infection of some new locality? To explain this by assuming that the majority of bugs soon disinfect themselves by a second feed is to close one's eyes to the more important factor in the spread of disease outside any given area. It does sometimes happen that an infected intermediate host is transported from one place to another, but this is a method of spread of disease which is quite subsidiary to that brought about by the wanderings of man himself.

On the Nomenclature of the Group Leishmania.

On the subject of the nomenclature of the parasites of Kala Azar and Oriental Sore, Patton is of opinion that as they conform to the herpetomonas type they should be known by the generic name of *Herpetomonas* and not *Leishmania*, and he makes the somewhat remarkable statement that "as the parasite of Sleeping Sickness passes one stage of its life-history in the blood and organs of man, and its multiplicative stage in an invertebrate *Glossina palpalis*, so in the same way the parasite of Indian Kala Azar passes one stage of its life-history in the blood and organs of man and its multiplicative stage in an invertebrate *Cimex rotundatus* : there would, however, be no justification for placing the parasite of Sleeping Sickness in any other genus but *Trypanosoma*." Now it is this very reason which, justifying the inclusion of the parasite of Sleeping Sickness in the genus *Trypanosoma*, supports the creation of a new genus *Leishmania*. The genus *Trypanosoma* includes flagellates which have both a vertebrate and invertebrate host, and the parasite of Kala Azar agrees with it in this ; but the genus *Herpetomonas* includes flagellates which have only one, and that an invertebrate host. The fact that the parasite of Kala Azar has two hosts and can live and multiply in the organs of a vertebrate shows it to be profoundly different from the true *Herpetomonas*, which lives only in the invertebrate. This fact alone would be sufficient to establish the genus *Leishmania*. It is admitted that the *Leishmania* resemble the *Herpetomonas* in their life-histories as far as we know them, but in any classification transition forms between the groups occur, as must be so from an evolution standpoint. When a *Herpetomonas* becomes so changed that it has acquired the power of living and developing as an intracellular parasite in the body of a warm-blooded animal, and giving rise to such a serious disease as Kala Azar, we are justified in concluding that it has passed out of the genus *Herpetomonas*, from which it originated, into the genus *Leishmania*.

EXPERIMENTS ON THE DEVELOPMENT OF *LEISHMANIA TROPICA* IN *CIMEX LECTULARIUS.*

In an earlier note I pointed out that I had produced in myself an Oriental Sore by inoculation, performed in Aleppo in August 1911. Six and a half months later the sores—three in number—appeared. They have increased in size, are of the non-ulcerating variety, and admirably suited for feeding experiments. Numbers of bed-bugs, *Cimex lectularius,* nearly all of which were hatched in the laboratory, were fed on the sore and kept in the cool incubator at 22°–23° C. for varying intervals before dissection. The conditions under which the experiments were conducted were ideal ones for giving good results. I had formerly shown that in Bagdad the *Leishmania tropica* would develop into flagellates in the bug (*Cimex rotundatus* ?).

I have now obtained proof that a similar development to flagellate forms will take place in *Cimex lectularius.* The development has been most disappointing, for flagellates were found in only a small percentage of individuals and then only in scanty numbers. Surely if the bug was the true host, some indication of active multiplication, ending in a heavy infection, must have occurred. The bugs were all fed upon the thin red epidermis over the sore. Patton has stated that bugs so fed cannot possibly dislodge parasites from the sore, but that they only obtain them from the blood circulating at the spot, as their proboscis is not long enough to reach the parasites in the sore. I have shown elsewhere that this view is improbable, since the skin over the infected macrophages may be hardly more than 50 μ in thickness and that the proboscis of a bug could readily penetrate so far. I have now obtained a better proof of this, for in the case of fleas feeding on the sore I have shown definitely that they take up free parasites while feeding and these in numbers too great to admit of their having come merely from the circulating blood. If the flea can do this, why not the bug? The further details of the flea experiments will be found below. The following experiments were conducted with *Cimex lectularius* :—

Number of bugs fed.	Mumber of days between feeding and dissection.	Number of bugs giving positive result.
26	9	0
14	8	0
29	7	0
10	6	0
12	4–6	0
5	3	1
9	2	2
Total...... 105		3

All these were young bugs which had their first feed upon the sore, except seven which were dissected after eight days and eleven of the twelve which were dissected after four to six days. It will be clear from the table that of the one hundred and five bugs which fed on the sore only three gave a positive result. In these three the number of flagellates was small, so that it is just possible that some of the others were infected and the flagellate forms overlooked. A further point of interest is the fact that those infected were dissected only two or three days after feeding, while none of the much larger number dissected later showed any such infection. If any active multiplication had taken place it is certain that I cannot have overlooked the infection. There is thus no evidence to show that the ingestion of a small number of leishmania will give rise to a massive infection. In my experiments conducted in Bagdad I found that one bug which was dissected twenty-four hours after feeding and three which were dissected forty-eight hours after, had become infected with flagellates. Patton found flagellate forms in bugs found upon the Cambay sore up to a much later date after feeding, and in one instance he claims to have found his post-flagellate forms in the bug.

The characters of the flagellates found in the *Cimex lectularius* do not differ in any way from those previously described by me. They resemble the forms met with in the artificial cultures. The flagellates usually occur in groups of three or four clustered together, indicating that such a group has probably developed from a single parasite. If the number of parasites ingested had been large as in Patton's Kala Azar experiments, then most certainly there would have been a corresponding number of rosettes of flagellates and one would have had a massive infection. It appears as if in the bug the development along the cultural lines proceeds to a certain extent and then comes to an end owing to failing nourishment; and just as in cultures which are old and in which the nutriment has been exhausted there appear round non-flagellate forms, so in the bug such forms are developed when the blood is nearing the completion of its digestion.

EXPERIMENTS CONDUCTED WITH FLEAS.

Owing to the fact that Basile claims to have transmitted the Mediterrenean Infantile Kala Azar by means of fleas, and, further, that he has described flagellate forms from these which are regarded as developmental forms of leishmania, I thought it would be of interest to investigate the behaviour of *Leishmania tropica* in these animals. Basile, Sangiorgi, and Alvarez and da Silva claim that flagellate forms found by them in fleas taken from dogs infected with Kala Azar represent the leishmania taken up; Basile further claims that these developmental forms appear in the fleas when they are fed upon spleen-juice of infected dogs. It would seem, then, that the evidence in favour of these contentions was conclusive; but quite recently Nöller, who has conducted experiments in the transmission of *Trypanosoma lewisi* by means of the dog-flea *Ctenocephalus canis*, has shown that quite a high percentage (12 per cent.) of these fleas may harbour a natural herpetomonas which resembles so closely the developmental forms of leishmania that he concludes that the flagellates observed in the fleas by Basile and others are really natural flagellates and not leishmania as they have supposed. In his recent report on Kala Azar, Patton has also come to a similar conclusion; and from the experiments about to be recorded, I can testify to the great difficulty of such experiments owing to the possibility of a natural herpetomonas in these fleas. Unless one most carefully excludes such a flagellate, there is nothing whatever to prevent an error of interpretation, for the forms of herpetomonas of the flea so closely resemble the leishmania that microscopical examination is hardly sufficient to separate the two.

Nöller's Method of handling Fleas for Experiment.

In Nöller's most interesting paper on the transmission of *Trypanosoma lewisi* by the dog-flea, there is described a method of experimentation with fleas which has rendered easy the handling of these elusive and active creatures. I have employed this method and find it most suitable and convenient; and for the sake of those who may wish to experiment with fleas and have no opportunity of consulting Nöller's original article, I will describe the method, which is really the securing of the fleas on fine wire, after the manner of those who exhibit aerobatic and so-called tame fleas at Fairs and similar Shows. Nöller recommends the use of fine silver wire, 0·15 mm. thick. I have used the finest copper wire from a galvanic coil and find it works equally well. A piece of this, about 10 cm. in length, is doubled into a loop and the two ends are held between the finger and thumb of the right hand while the loop is held over a needle so that the wire is kept tight. By movement of the finger and thumb the wire is twisted till only a very fine loop is left at the end through which the needle passes. The exact size of the loop is rather an important matter, but it varies with the size of the flea to be used Experience alone can act as a guide. The flea to be secured—and for first practice the large dog-flea is most convenient—is dropped on to the surface of water in a dish. It is then picked out with the finger and thumb of the left hand and held in such a way by the abdomen that the head is directed upwards and its dorsum towards oneself. By gently rolling the finger and thumb the head of the flea can be made to protrude while the abdomen is still held. The loop of wire is then taken in the right hand and carefully slipped over the flea's head. If the loop is of the correct size it will pass easily over the head but not back on to the abdomen. It will be seen that the flea commences to make violent struggles and finally it will manage to get its first pair of legs through the loop. Now is the critical moment. The flea is held only by the wire and with a fine pair of forceps the loop is pinched up smaller behind the dorsum of the flea. If secured in this way the flea cannot escape and will live quite well in cotton-wool for over two months. I have kept one flea alive in this way for two months and a quarter, and it was an unfortunate neglect of feeding that led to its untimely death. Those who have to use fleas in experimental work are much indebted to Nöller for introducing this simple method of handling them. With the wire bent in a suitable manner in two planes it will rest on the skin at any spot, and the flea will feed apparently in perfect comfort.

The Feeding of the Fleas.

Nöller has described the feeding of these fleas. After sucking blood for about ten minutes—it may be more or less than this—fæces are passed, and usually these are ejected with considerable force, so much so that they may fall on the skin as much as an inch away from the flea. The first fæces passed consists of digested blood, but even with this first faeces there is admixture with fresh blood, as is shown by the fact that live *Trypanosoma lewisi* may be passed in the first fæces when the flea is feeding on an infected animal. After the passage of faeces the fleas continue feeding and may pass fæces which eventually becomes pure blood as frequently as every half-minute. If the flea is observed by transmitted light with a hand-lens during the feeding act, it is noticed that the gut is in a state of most violent peristaltic contractions, first in one direction and then in the other, so that the whole contents of the intestine are mixed together. It is this fact that accounts for the appearance of fresh blood in the first fæces passed.

It is a simple matter while the flea is feeding to collect the ejected fæces on cover-glasses held in forceps in a suitable position to receive them. The fæces can be made into films and stained. In this way it is easy to determine if a flea is infected with flagellates or not. The peristaltic action is so violent that if flagellates are present, and even if they are attached to the lining of the gut, some at any rate would be expelled. Thus in conducting experiments with leishmania it can readily be determined if a flea is clean by feeding it upon some uninfected person two or three times, and examining the fæces passed during these feeds. From a practical experience of this method I can testify to its accuracy, for those fleas which have been found naturally infected with herpetomonas or *Trypanosoma lewisi* have remained infected during the whole time that they have lived and fed upon human blood, while none of those which were not infected at the first feed have acquired any such infection unless fed upon infected blood.

The Fæces passed by Fleas when Feeding on the Sore may contain Leishmania.

At the outset of my experiments with fleas and *Leishmania tropica* I found that fleas while feeding upon the sore passed fæces containing these parasites. It was evident that the fleas had taken them up directly from the sore and had dislodged them from the infected macrophages. The parasites are always free and not included in leucocytes, as they would be if they came from the peripheral circulation. I have noted the presence of these leishmania in the fæces in nearly every instance (upwards of 50 feeds) that a flea has fed upon the sore, so that there is no possibility of a confusion with any herpetomonas of the flea. The fleas, moreover, had given negative results when fed upon uninfected persons. It can thus be fairly safely concluded that in almost every instance that a flea fed upon this particular Oriental Sore—which, as I have said, was one peculiarly suited to such experiments—leishmania were taken up, as proved by examination of the fæces passed during the act of feeding.

The Leishmania do not Develop in the Flea.

In order to determine if any development would take place, the following procedure was adopted :—The clean flea was fed upon the sore and its fæces examined for leishmania. It was then kept in the cool incubator (22°–23° C.) for a variable time. It was then fed again, either upon the sore or upon my arm below the sore. The fæces were collected again and examined. When the feed was upon the sore, leishmania were again found to have been taken up, whereas in no case were they seen when the feed was on the skin below the sore, however near the sore that might be. So that the evidence is again in favour of the fleas having dislodged the parasites from the sore. This process of feeding on the sore at varying intervals, with the examination of the fæces passed during the feed, would give some indication of flagellates in the gut if any development of the leishmania had taken place. In fleas naturally infected with herpetomonas or *Trypanosoma lewisi* there is no difficulty in finding these in the fæces. Yet with the leishmania I never on any occasion found the least indication of a development into flagellates, for the leishmania taken up at one feed had completely disappeared before the next. In a certain number of instances these observations were controlled by dissection of the fleas, but this gave no better result. So that I have obtained no evidence that *Leishmania tropica* will develop in the dog or human flea (*C. canis* and *P. irritans*), both of which were used.

Description of Experiments.

1. A *Ctenocephalus canis* was fed upon the sore twelve times and once upon the skin near it between July 29 and August 12. On nearly every occasion when it fed from the sore, leishmania were found in the fæces. The intervals between the feeds varied from one to eight days, and on no occasion was any development noted. On September 15 the flea was fed on a rat infected with *Trypanosoma lewisi* and again on a clean rat on Sept. 21, 24, and 26. On none of these occasions were developmental forms of *T. lewisi* seen in the fæces. On Sept. 27 it was again fed on the *T. lewisi* rat. On Oct. 3 it fed on the clean rat and the fæces contained the typical small trypanosomes which are characteristic of the *T. lewisi* infection of the flea. On Oct. 8 the flea was found dead. Though it did not become infected with leishmania, it became infected with *T. lewisi*.

2. A *C. canis* was fed on the sore six times and once on the skin near the sore between Aug. 3 and 31, when it was dissected. No indication of development was found either in the fæces or on dissection.

3. A *Pulex irritans* was fed upon the sore on Aug. 3 and 10. It was dissected with negative result on Aug. 13.

4. A *C. canis* was fed upon the sore on Aug. 10, 13, and 16. Fæces always negative as regards development. Dissected on Aug. 19 with negative result.

5. A *C. canis* fed on sore on Aug. 19 was dissected on Aug. 26—negative.

6. A *C. canis* was fed on sore on Aug. 24 and dissected Aug. 26—negative.

7. A *C. canis* fed on skin below sore on Sept. 6—fæces negative. On Sept. 12 fed on sore and on Sept. 15 on the *T. lewisi* rat. On both these occasions fæces contained no developmental forms of leishmania. On Sept. 21 flea fed on my arm—fæces negative ; and again on Sept. 24 it had a small feed. On Sept. 25 flea fed on my arm and passed fæces containing typical small *T. lewisi* trypanosomes. It again fed on human blood on Sept. 28 and Oct. 1 : on both occasions small trypanosomes were in the fæces. On Oct. 4 flea fed on a clean rat, while the fæces passed were collected on rat's hair and allowed to dry for one hour. It was then placed in mouth of another rat which did not become infected with *T. lewisi*. On Oct. 9 flea died and was found to have large infection of small trypanosomes in hind gut. Some of the fæces which were passed on Sept. 28 were placed without drying into the mouth of a clean rat. The rat was infected with *T. lewisi* six days later. This is a confirmation of Nöller's results. The clean rat upon which this flea fed was the same as that upon which the first flea fed after it had taken up *T. lewisi*. It was not contaminated with flea fæces and has not become infected.

8. A *C. canis* was fed on the skin of my arm on Sept. 2 and on the sore on Sept. 6. Fæces negative. Flea was dead on Sept. 12.

9. A *P. irritans* was fed on the *T. lewisi* rat on Sept. 21 and dissected on Sept. 25. It was found to contain the developmental forms of *T. lewisi*, so

that this flea, as well as the dog-flea, will probably prove to be a transmitter of *T. lewisi.*

10. A *C. canis* was fed on the sore on Oct. 3, 4, 8, 14, 21. On no occasion was there any developmental form in the fæces.

11. A *C. canis* was found, when fed on clean human blood, to be infected with the small *T. lewisi* trypanosomes. It must have previously fed on an infected rat. It was fed on the sore on Oct. 8 and Oct. 14. In fæces only unaltered leishmania and small *T. lewisi* trypanosomes.

12. A *P. irritans* was found to be infected with herpetomonas * at its first feed. From Aug. 24 to Nov. 4, when it died, it had had sixteen feeds of human blood and had constantly passed large numbers of herpetomonas, both flagellate and non-flagellate, in its fæces. These resemble very closely developmental forms of leishmania.

With the fæces of this flea collected on sterile cover-glasses cultures have been obtained on NNN medium as follows :—

1. Fresh fæces very rich culture.
2. Fæces allowed to dry for one hour at laboratory temperature in sterile Petri dish gave a culture after a few days.
3. Fæces dried for four hours gave only a growth of sarcinæ.
4. Fæces dried for two hours and a half gave growth of flagellates.
5. Fæces spread on cover-glass and dried for 24 hours gave culture of flagellates.

It is thus seen that the cystic forms passed in the fæces are very resistant and will stand a considerable amount of drying. Further, the fæces passed by the flea are, as a rule, sterile as regards bacteria which will grow upon the NNN medium. The cultures of herpetomonas are exceedingly vigorous and have now been maintained for several generations, so that they will probably prove to be culturable for an indefinite period.

13. A *C. canis* from a cat was found to be infected with herpetomonas on its first feed.

Conclusions drawn from these Experiments.

It is seen from these experiments :—

1. That the dog and human fleas, *Ctenocephalus canis* and *Pulex irritans*, readily take up *Leishmania tropica* from the sore and that some of them are passed in the fæces voided during the feeding act.
2. That if a flea is infected with flagellates the fæces voided during the feed will contain some of these.
3. That the leishmania taken up do not develop into flagellate forms to produce an infection of the flea's gut.
4. The fleas that have not become infected with leishmania may be infected with *T. lewisi.*
5. When once infected with herpetomonas or *Trypanosoma lewisi* the fleas remain so infected for long periods whether they feed upon human or rat's blood. The human blood appears to be quite favourable to the development of *Trypanosoma lewisi* in the flea.

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* This flagellate of the human flea is quite distinct from the crithidium described by Porter from this flea. It is a true herpetomonas and has never shown any crithidial forms. After its death the flea was dissected and was found to contain enormous numbers of herpetomonas in all stages of development. (A. Porter, 'Parasitology,' 1911, pp. 237-254.)

6. *Trypanosoma lewisi* appears to undergo its development in the *Pulex irritans* as well as in the dog-flea *Ctenocephalus canis*.
7. As Nöller has shown, it is the fæces passed by the flea which, when licked up by the rat, produce an infection. The act of feeding itself apparently does not infect. This has been confirmed by feeding the dog-flea on a *T. lewisi* rat, feeding it in future on a second rat, and giving the fæces passed during the feed to a third rat. It is only the third rat that becomes infected. The incubation period was six days in the rat.
8. The small oval encysted forms of the herpetomonas passed by the flea are very resistant structures. They will withstand drying for 24 hours, and then give rise to a culture of herpetomonas when introduced into NNN medium.

The Bearing of these Results on Basile's Experiments.

The failure to obtain any development of *Leishmania tropica* in the dog or human flea and the fact that the flea may contain a natural herpetomonas almost indistinguishable from cultures of leishmania suggest to my mind that Basile and those who have followed him may have misinterpreted what they found in the fleas. Basile gave no proof that he had taken care to exclude a natural flea infection, and, as a matter of fact, the number of fleas found by him to be infected with what he interpreted as leishmania was well below the percentage found in other places to be naturally infected with herpetomonas. Comparing the herpetomonas found by me in the fleas I find that they correspond entirely with those described by Basile and Alvarez and Pereira da Silva.

It may be urged that my flea experiments were conducted with *Leishmania tropica* and Basile's with the leishmania of Infantile Kala Azar and that the results are not comparable. The leishmania derived from all sources behave so similarly in culture media and in bed-bugs that if the one develops to any extent in such a host as the flea it would be expected that the other would at least show some indication of such a development. Still I recognise this difference, and only trust that now that Nöller has shown us how fleas can be conveniently handled for accurate experiment, Basile or some other observer will repeat the experiments with every precaution to exclude any error.

I make no comment on the actual transmission experiments carried out by Basile in transmitting Kala Azar to dogs by means of fleas*. Experiments conducted in the Sudan by Marshall with infected dogs and fleas have given only negative results. I am quite in accord with Nicolle and Basile, who maintain that the Kala Azar of the Mediterranean districts is the same disease in children and dogs. Patton finds himself in complete agreement with Gabbi, who regards the infantile disease as distinct from the canine. It seems to me that the established fact that in all the endemic centres for Infantile Kala Azar along the Mediterranean there canine Kala Azar exists, is in itself almost convincing. There are such cases as that recorded by the Sergents, Lombard, and Quilichini, where a child, a dog, and a cat were found to be infected with Kala Azar in an isolated farm near Algiers, which are difficult to explain on any other hypothesis. In addition we have the experimental evidence of the possibility of producing Kala Azar in dogs by inoculation of the human virus. Surely it is a greater assumption to conclude under these circumstances that the canine disease is distinct from the human than that they are one.

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* Quite recently, in the 'Bulletin de la Société de Pathologie Exotique' of Oct. 9, 1912, the brothers Sergent, Lhéritier, and Lemaire have described the successful transmission of Canine Kala Azar from dog to dog by means of the flea *Ctenocephalus canis*. It is unfortunate, from the point of view of the experiment, that the dog which was found to have become infected never showed any parasites in its liver, for it was by liver puncture performed before the experiment that the dog was supposed to be free from infection. After infection was proved by examination of the spleen and bone-marrow, both microscopically and by culture, the liver still gave negative results. Flagellates were found in the fleas taken from the infected dog and also in others from uninfected animals, but the authors have not proved that they were developed from leishmania taken up by the fleas.

The distribution of the Mediterranean Kala Azar in children and dogs points to the flea as a likely transmitter of the malady. Basile claims to have proved this, but Franchini's recent results, in which he has found that the leishmania taken up from spleen puncture material develop into flagellates in these insects' guts, opens another possibility, for mosquitoes as well as fleas feed upon both dog and man. In India it is quite possible that the bed-bug is the carrier of the disease. It is an insect which has been blamed for the transmission of many maladies, but has been clearly proved to transmit none. At present it is still unconvicted' for I fail to find in the fact that the leishmania will develop in its gut even to Patton's post-flagellate stages any conclusive evidence that it is the true host and natural bearer of this fatal malady. Till the experiment of transmission has been carried out we must be content to keep an open mind upon this subject.

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Experimental Amœbic Dysentery and Liver-Abscess in Cats.

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Early Experiments on the Production of Amœbic Dysentery in Animals.

Hlava and Kartulis were the first to show that amœbic dysentery could be produced in cats by the injection per rectum of the mucus or fæces from cases of amœbic dysentery in man. This experiment was repeated by Quincki, Roos, Kruse, Pasquale, Strong, Musgrave, and others, while Kartulis further showed that dogs could similarly be infected, and Musgrave and Clegg produced dysentery in monkeys in the same manner. In experiments of this kind there is always the possibility that other pathogenic organisms besides the amœbæ are injected and that they may be the real cause of the dysentery. Kruse and Pasquale were successful in producing dysentery in cats by the injection of pus from an amœbic liver-abscess which was shown to be bacteria free. As Hartmann has pointed out, the entire absence of bacteria in a liver-abscess is very difficult to demonstrate. All that one can say is that the cultural methods employed failed to demonstrate their presence. It is always possible that some other method of culture not employed would have succeeded. Jurgens stated that the amœbæ actually forced their way through the healthy epithelium of the cat's gut, and Schaudinn attributed their power of doing this to their tough ectoplasmic pseudopodia. It was Schaudinn who first clearly distinguished a non-pathogenic and pathogenic amœba of man under the names of *Entamœba coli* and *Entamœba histolytica.* By feeding cats on material containing cysts of *Entamœba histolytica* amœbic dysentery was produced.

Is E. tetragena *the same as* E. histolytica ?

Viereck examined an amœba from cases of dysentery and discovered that it differed in its life-history from Schaudinn's *E. histolytica.* He accordingly named it *E. tetragena*, from the fact that the mature cyst contains four nuclei. The cysts of *E. tetragena* were then recognised by Hartmann and have now been found in almost every part of the world where amœbic dysentery occurs. The amœba of Viereck has come to be looked upon as the common pathogenic amœba of man. It is thus difficult to account for Schaudinn's *E. histolytica.* Hartmann has re-examined many of Schaudinn's preparations and finds that his *E. histolytica*, in most cases at least, corresponds with *E. tetragena* of Viereck. It seems highly probable that the pathogenic amœba with which Schaudinn worked was the common pathogenic form and that he failed to recognise the cysts of Viereck with their four nuclei and that the small spores he described were other structures unconnected with this amœba. If this position is adopted it comes about that the amœba of Viereck, *E. tetragena*, is none other than *E. histolytica* of Schaudinn and that Viereck regarded it as a distinct species because he had discovered the true life-history, which, of course, differed from the erroneous one given by Schaudinn. Hence, provided Schaudinn was right in naming the pathogenic amœba *E. histolytica*, the name *E. tetragena* is no longer required. The common pathogenic amœba of man is still *E. histolytica*, but the life-history is not that described by Schaudinn, but that discovered by Viereck.

Attempts to confirm Schaudinn's Observations on E. histolytica.

During a stay in Khartoum in 1907, I studied cases of amœbic dysentery in the Military Hospital, but I failed to discover any indication of the development described by Schaudinn for *Entamœba histolytica* (Third Report of Wellcome Research Laboratories. p. 129). Since that time I have repeatedly tried to obtain some confirmation of this development, especially as regards the production of the small infective tough-capsuled spores which Schaudinn supposed this amœba to produce. My endeavours in this direction were always unsuccessful, so that I came to doubt whether Schaudinn had really described the true development. Viereck's discovery has apparently cleared the ground, for we now know that the human pathogenic amœba produces not the tough spores of Schaudinn but the transparent cysts which Viereck first described. Craig's confirmation of Schaudinn's results are hardly convincing, on account of the doubtful technique he employed.

Out of a large number of observations on cases of amœbic dysentery in man, only on six distinct occasions have I encountered the typical cysts of the pathogenic amœba in cases of dysentery. It is unfortunate for diagnostic purposes that the cystic forms occur only in a small percentage of cases of amœbic dysentery, for they are comparatively easy to recognise; they can readily be distinguished from the cysts of *Entamœba coli*, and it is their presence which assures one more than anything else that the amœba in question is the pathogenic form. It is admitted that the pathogenic and non-pathogenic amœbæ of man are subject to much variation, so that at one time the differences may be most marked, while at others they are hardly to be recognised. What it is that causes the amœbæ to encyst is not known. As a rule, during the height of a dysenteric attack encysted forms are not seen, but they appear to be formed when the patient is recovering naturally from the disease. So that encysted forms of the pathogenic amœbæ are seen most usually during periods when the dysenteric process is abating in its activities. Another feature of this cyst-formation is that when they occur the cysts usually do so in very large numbers, so that at such times one encounters more encysted amœbæ than free ones. They may be so numerous that three or more occur in every field of the Zeiss $\frac{1}{12}$ objective in thin cover-glass preparations, and by suitable methods very beautiful preparations of the encystment process can be obtained. From such I have been able to confirm in every essential respect the observations of Viereck and Hartmann on the pathogenic amœba, but I have not been able to obtain any confirmation of the life-history of the pathogenic amœba as Schaudinn described it.

The Production of Amœbic Dysentery in Cats.

The experiments to be described in this paper were conducted with material obtained from a case in a European from Bombay in which numbers of amœbæ, both free and encysted, occurred. It is interesting to note that the case was admitted to the Branch Hospital of the Seaman's Hospital at the Royal Albert Dock, not for dysentery but for severe anæmia. An examination of the stool, however, showed that there was a certain amount of blood-stained mucus present in the form of small flakes, while a closer inspection with the microscope revealed a large infection of free and encysted amœbæ, which from the typical character of the cysts was seen to be the pathogenic form (Viereck's *E. tetragena*). About 5 c.c. of this material was injected per œsophagus and per rectum into two cats. Both of these contracted amœbic dysentery. Such experiments have been conducted with this amœba by Viereck, Werner, Hartmann, and more recently by Darling in Panama. It has been shown by these observers that ingestion of the amœbæ alone by the cats does not produce dysentery, whereas ingestion of the encysted forms will do so. Injection of the amœbæ themselves per rectum will produce dysentery. In my experiment the first two cats were injected both per œsophagus and per rectum. The method of injection was as follows:—The terminal six inches of a No. 6 soft catheter was fixed to the end of a glass syringe. The catheter was passed up into the rectum or down the œsophagus into the stomach and about 5 c.c. of material injected. Both these cats were passing typical dysenteric stools containing much blood and mucus and many amœbæ nineteen days after the injection. It is probable that they had already been infected for some days, so that the incubation was less than nineteen days. From these cats others were infected, and I have been able to carry on the infection through four passages, and but for an accident could have carried it much further, as there was no sign of decreasing virulence in the strain of amœbæ employed.

Hartmann states that from his own experiments with this amœba and those of Viereck and Werner the infection of cats is not satisfactory, and that after two or at most three passages the infection ceases. From my experiments it seems clear that with care it would be possible to carry on the infection indefinitely in cats. In the sub-inoculations the ulcerated large intestine of the cat (which had died naturally from the disease or had been killed when about to die) was opened and washed in physiological saline solution, which was then injected into the large intestine of the next cat. In only one instance did this fail to produce amœbic dysentery in the cat. The cat which did not contract dysentery was the largest used, so that to ensure success one must employ young kittens soon after they have become independent of the mother.

The Production of Amœbic Abscess of the Liver in Cats.

Apart from the fact that I have been successful in carrying on the infection in cats through more passages than have hitherto been done, a still greater interest attaches to other observations. In the first place, it is often seen that the mesenteric glands draining the ulcerated areas of the large intestine (it is nearly always the lower part of the large intestine which is ulcerated) are swollen, and I have frequently found that on section these contain a large number of amœbæ which appear to be feeding on the gland-tissue, for their bodies are often packed with gland-cells. This may result in the complete destruction of the glandular tissue. In the second place, Cat IX., which contracted dysentery as a result of the injection of amœbæ from the cat of the third passage, not only developed typical dysenteric ulceration of the gut, but also abscess of the liver, in which numerous amœbæ occurred. In all there were four superficial abscesses, the largest of which had a diameter of half a centimetre. Unfortunately the cat died of its dysentery; otherwise the abscesses would have increased in size, and it might have been possible to conduct further experiments with the amœbæ from them. It is perhaps necessary to mention that the liver of this cat had not been injured in any way, but the abscesses had developed spontaneously as a result of the amœbic infection. I have thus been successful in producing the whole picture of amœbic dysentery of man experimentally in the cat with its final complication of amœbic abscess of the liver. The fact that it was in the fourth passage that the abscesses developed indicates that the amœbæ had lost none of their virulence in these passages, and it points in a striking manner to the true pathogenicity of this amœba, viz. Viereck's *Entamœba tetragena*, which, as I have already said, is probably identical with Schaudinn's *Entamœba histolytica*.

The Invasion of the Tissues by the Amœbæ.

Exactly how the amœbæ find their way from the intestine to the liver is difficult to decide. It would most probably be by way of the portal blood-vessels; and this is perhaps borne out by the fact that Cat VII. developed pyæmia in which the whole blood-system contained pus. I have already pointed out that the amœbæ commonly invade the mesenteric glands, so that a lymphatic course is possible.

In sections of the ulcerated gut it is easy to study the invasion process. The amœbæ make their way to the bottom of the tubular gland in the large intestine. There they multiply and by pressure of their numbers or by the exertions of their pseudopodia, and probably through some toxic substance excreted by them, the lining cells are weakened and separated and the amœbæ pass into the connective tissue beneath. The epithelial cells commence to degenerate and the earliest stage of ulceration is reached. Bacteria are thus admitted and the destruction becomes more rapid, while the amœbæ push on to the deeper layers and give rise to the characteristic undermined ulcers of amœbic dysentery. In the case of the natural infection of mice with *Entamœba muris* I have shown elsewhere * that these amœbæ often enter the tubular glands of the large intestine of these animals and may make their way to the deepest extensions of these. In the case of this harmless amœba the invasion of the glands is not followed by any disintegration of the glandular epithelium, whereas the similar invasion of the glands of the cat by the human pathogenic amœba is immediately followed by dysenteric ulceration. What is it that causes this destruction? It seems to me that in the case of the pathogenic form there must be some toxic substance which so weakens the epithelium of the glands that the amœbæ are able to force their way through perhaps before it has actually disintegrated. I can hardly believe that the pathogenic amœba can force its way through the uninjured and healthy epithelium. If so, why does not *Entamœba muris* penetrate the glandular epithelium of the mouse's gut? To state that the pathogenic amœba has a tougher pseudopodium for this purpose is a pure assumption, for *Entamœba muris* may have pseudopodia which, as far as one can judge, are quite as substantial as those of the pathogenic forms.

Experiments with other Amœbæ from the Human Intestine.

In addition to experiments with the pathogenic amœba, I have attempted to infect cats with the harmless *Entamœba coli*. In the case of Cat VIII. there was administered per œsophagus and per rectum a large dose of fæces containing numerous free and encysted *Entamœba coli*, while Cat IX. received per rectum an injection of fæces from another case in which only free forms of *E. coli* occurred. Neither of these cats became infected, nor did they suffer in any way from the injections. They were later infected with material from dysenteric cats and they both contracted amœbic dysentery.

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* "Observations on the Protozoa in the Intestine of Mice," Archiv für Protistenkunde, Supplement I.

An attempt was also made to infect Cat V. with a small amœba which occurred in enormous numbers in the fæces of a native of India. The amœba in question varied in size from 3–10 μ and had a nucleus of the typical amœba limax type. It was constantly present in the stool during the three weeks through which the case was kept under observation. The amœbæ, though occurring in such numbers that as many as a dozen or twenty were found in each field of the Zeiss $\frac{1}{12}$ objective, appeared to have not the least ill-effect upon their host, who was admitted to the hospital for quite other reasons. The Cat V., though it received per rectum many millions of this amœba, did not become infected.

The Amœbæ as they occur in Cats.

There is no need to enter here into a detailed description of the pathogenic amœbæ as they occur in the cat. The normal healthy amœbæ in the cat correspond exactly with those from the human intestine, and just as in cases of amœbic dysentery in man many of the amœbæ are degenerate and show abnormalities in nuclear structure, so in the cat one encounters many altered forms. It is certainly correct, as Hartmann has shown, that in the cat the number of altered or abnormal amœbæ is greater than in man, who is a more suitable host. The occurrence of these abnormal forms, especially those in which the nucleus has undergone a change and has broken up into scattered chromidia, may easily lead to errors of observation. Hartmann admits that he himself was misled by such altered forms and that he saw in them a confirmation of Schaudinn's development of *Entamœba histolytica.*

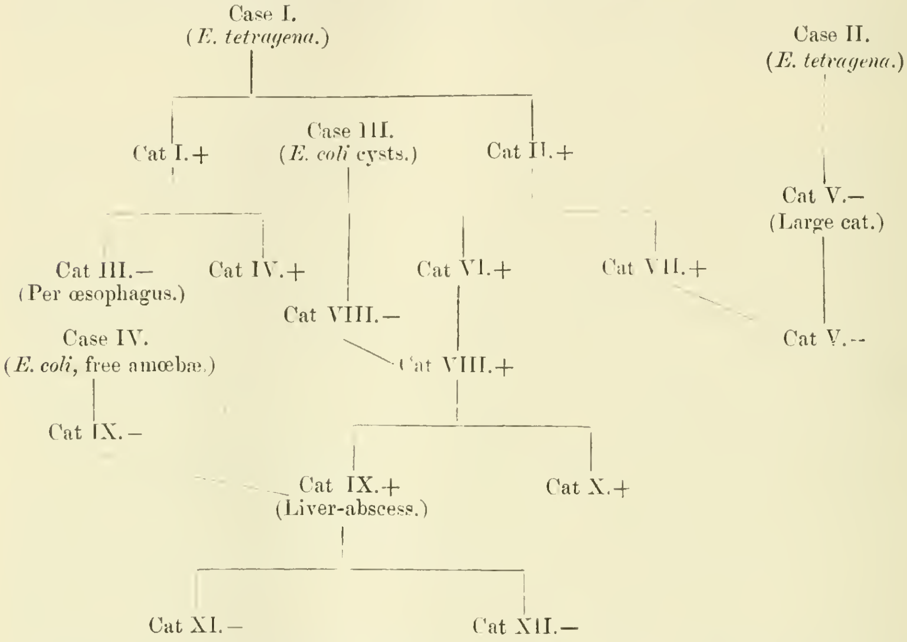
Another point which requires mention in connection with the experimental amœbic dysentery of cats is the absence of the encysted forms. On no occasion have I been able to discover the cysts of Viereck in the fæces of the cats. This is probably to be accounted for by the fact that the dysenteric process in the cats does not pass beyond the acute stage. In man cysts are rarely seen in the height of a dysenteric attack. Should the cats suffer from the acute dysentery and then commence to recover in a natural manner, it is possible that the encysted forms of the amœbæ would occur in the fæces.

Concluding Remarks.

As a result of these experiments it is seen that the human pathogenic amœba (*E. histolytica* of Schaudinn and *E. tetragena* of Viereck) when inoculated per rectum into cats is able to produce a typical ulcerative condition of the large intestine, of which the amœba is evidently the exciting cause. The fact that the infection may be carried on for four passages, and most probably indefinitely, in cats is further support of the pathogenicity of these organisms. Finally, the fact that after three passages the amœba occurred in abscess of the liver which had developed spontaneously apparently as a result of the amœbic infection is the last step in the argument. It is only in a small percentage of cases of amœbic dysentery in man that amœbic abscess develops, and so in cats it was in only one out of the nine which contracted amœbic dysentery that showed the complication of amœbic hepatic abscess. From the result of these experiments it seems hardly possible to doubt that the amœba is the true and exciting cause of amœbic dysentery and liver-abscess in man. The production of amœbic dysentery in cats has been accomplished before, but this is the first time that the final proof of the pathogenicity of the amœba has been obtained in the production of liver-abscess as it occurs in man as a complication of this disease.

The experiments with *Entamœba coli* have given negative results, though the number of amœba used in the experiments was enormous. It is a further proof that there occur in man both pathogenic and non-pathogenic amœbæ.

Tree indicating the various passages of E. tetragena *in the Cat.*



Cats I., II., and VIII. were injected per œsophagus and per rectum, Cat III. per œsophagus only, and the rest only per rectum. The material was obtained from four cases, two of which were dysenteric with *E. tetragena*, and two intestinally healthy and harbouring *E. coli*.

Details of the Cats used in the Experiments.

Cat I.—July 10. Injected per rectum and per œsophagus with amœbic fæces from human case.—July 29. Typical dysenteric stool with blood mucus and many amœbæ.—August 9. Cat died. Ulceration of the large intestine and cæcum and innumerable amœbæ in the large intestine. No amœbæ above the cæcum. Mesenteric glands swollen. Liver normal.

Cat II.—July 10. Injected as Cat I. and was in a condition similar to that of Cat I. on July 29. This cat continued to pass blood and mucus with amœbæ up to August 27, when, owing to its bad condition, it was killed. Ulceration was limited to the lower part of the large intestine. The liver was normal. The mesenteric glands were swollen.

Cat III.—On August 9 the cat was injected into the stomach per œsophagus with saline washings of the large intestine of Cat I. Innumerable amœbæ were present. Cat died on August 18 (cause not known). There had been no sign of dysentery nor any amœbæ in the fæces. There was no ulceration of the large intestine.

Cat IV.—On August 9 injected per rectum with saline washings from Cat I. On August 14 cat was passing quantities of blood and mucus and many amœbæ.—August 16. Amœbæ exceedingly numerous in dysenteric fæces. On the next day this cat escaped from its cage and was not recovered.

Cat V.—On August 15 this cat, which was a large one, was injected per rectum with dysenteric stool, in which amœbæ occurred but no cysts, from another human case. This cat did not develop dysentery, so on September 5 it was re-inoculated with saline washings of large intestine of Cat VII., which had been injected from Cat II. It did not develop dysentery. This is the only cat that has proved refractory to inoculation.

Cat VI.—On August 6 was inoculated per rectum with the stool of a man in which there were innumerable small amœbæ of the limax type. The man had no sign of dysentery. The cat never showed any infection with these small amœbæ, so was re-inoculated on August 27 with saline washings of large intestine of Cat II.—August 30. Cat was passing blood and mucus and amœbæ.—September 8. Cat was found dead. There was ulceration of the large intestine and innumerable amœbæ in the fæces. Contents of large intestine washed out in saline and injected into Cat VIII.

Cat VII.—On August 21 was injected per rectum with material obtained by washing out large intestine of Cat II. with saline enema. Cat did not appear to be developing dysentery, so was re-injected per rectum on August 27 with saline washings of large intestine of Cat II. which had been killed on this day.—August 30. Active amœbæ in stool.—September 2. Passing quantities of blood and mucus and amœbæ.—September 5. Cat was killed, as it was in bad condition. The organs appeared pale and on cutting exuded blood and pus. All the vesseis contained the same pinkish fluid, which was found to be a mixture of blood and pus. The cat evidently had pyæmia. There were no abscesses in the organs. The large intestine was very much ulcerated and contained innumerable amœbæ. The saline washings were injected into Cat V., which did not become infected.

Cat VIII.—On August 21 was injected per rectum and per œsophagus with a stool containing innumerable cysts of *Entamœba coli* and active amœbæ, which were presumably also *E. coli.* The case had no sign of dysentery. Up to September 8 there had been no indication of any infection and no amœbæ had appeared in the stool. On this date the cat was injected per rectum with saline washings of large intestine of Cat VI. (found dead September 8—innumerable active amœbæ).—September 10. Blood and mucus in the stool.—September 26. Cat was very ill after having continuously passed blood, mucus, and amœbæ since it was first infected. It was killed and saline washings of large intestine were injected into Cats IX. and X. There was typical ulceration of the large intestine with enlargement of the mesenteric glands.

Cat IX.—On August 29 was injected per rectum with about 10 c.c. of a stool containing numerous active amœbæ which were presumably *Entamœba coli.* No cysts were seen. The case had no sign of dysentery. Up to September 26 there had been no indication of any infection in the cat nor had amœbæ been found in its fæces. The cat was accordingly injected per rectum with saline washings from intestine of Cat VIII. On September 30 the cat was passing blood and mucus. October 5, the cat was found dead. There was much ulceration of the large intestine, the mesenteric glands were swollen, and there were four abscesses in the liver. Saline washings of large intestine injected into Cats XI. and XII. The abscesses of the liver contained many amœbæ.

Cat X.—On September 19 was injected per rectum with some mucus passed by Cat VIII. It contracted a mild attack of dysentery, passing small quantities of blood and mucus with not many amœbæ. It was re-injected per rectum on September 26 with saline washings from Cat VIII. On September 28 it was found to be suffering from a virulent diarrhœa with very liquid stools. It was accordingly killed. No amœbæ were found in the gut, but there was one small ulcer which was found to be amœbic in nature when sectioned. The diarrhœa which attacked this cat was also responsible for the death of another cat of the same litter which had not been injected.

Cat XI.—On October 5 was injected per rectum with saline washings from Cat IX. which had died in the night. Most of the amœbæ in Cat IX. were dead at the time of the injection. The cat did not become infected and died of virulent diarrhœa on October 11. There was no ulceration of the gut.

Cat XII.—Injected on the same day, but did not become infected. Died on October 14 of diarrhœa, like Cat XI. There was no ulceration of the gut. The strain of amœbæ was lost, owing to the fact that Cat IX. had died unexpectedly in the night.

NOTES ON TRYPANOSOMA RHODESIENSE FROM THREE CASES OF HUMAN TRYPANOSOMIASIS.

By C. M. WENYON, M.B., B.S., B.Sc.,
Protozoologist to the London School of Tropical Medicine,

and

H. M. HANSCHELL, M.R.C.S., D.T.M. & H. (Camb.),
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The three cases (G, E, and T) from which the trypanosomes were obtained are the subject of a paper by Dr. Daniels in another part of the present journal. Observations were made on the parasites as they occurred in man and also in rats. Though the trypanosomes were fairly numerous in one of the cases, none of those examined showed the posterior arrangement of the nucleus supposed to be characteristic of the *Trypanosoma rhodesiense.* These forms, as Stephens and Fantham first noted in the strain with which they worked, appeared first in the blood of rats inoculated from man. The appearance of these forms was supposed by them to be distinctive of this trypanosome, but we now know that similar forms may occur in other trypanosomes—*T. pecaudi*, *T. equiperdum*, and *T. brucei.*

The "posterior nuclear forms" * occurred in the first rat inoculated by us from each of the cases, so that we may assume that the three strains were truly *T. rhodesiense.* But though the posterior nuclear forms occurred in each case, the percentage of these forms varied very much. Thus the G strain in the first rat inoculated by us showed a percentage varying from 0–·9, the E strain a percentage of 3–7, while the T strain contained a great percentage, varying from 13–40. In each case the percentages were based on counts of one thousand trypanosomes made every three days. Taking, however, the observations made on several rats through which the strains of trypanosome have been passed, it is found that in the G strain the percentage of posterior nuclear forms may vary from 0–·9, as in the first rat inoculated, in the E strain from 6–72, where the percentage was much higher in one rat than in the others, while in the T strain the percentage varied from 13–40 as in the first rat. Considering the results generally, it is found that the G strain may contain none or very few posterior nuclear forms, while in the T strain the posterior nuclear forms may constitute at least one-third of the trypanosomes present. The E strain occupies an intermediate position. It is not proposed to give details of these results. These will be reserved for a subsequent work, but it is quite clear that the percentage of these forms may vary within very wide limits. It has been found that occasionally the G strain shows no posterior nuclear forms. In such cases it would be difficult to separate the trypanosome from *T. gambiense*. With variations within such wide limits it is conceivable that a strain might arise in which no posterior nuclear forms occurred in the various passages through the rats. Would such a trypanosome be *T. rhodesiense* or *T. gambiense*? In this respect it certainly seems as if the two trypanosomes merge into one another.

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* All those trypanosomes which show a decided displacement of the nucleus towards the posterior end of the body are included. In nearly every instance the nucleus lies either against the kinetonucleus or separated from it only by the vacuole. Though this nuclear displacement occurs most commonly in the broad trypanosomes with a short free flagellum this is by no means always the case, as others of quite normal dimensions show this peculiarity, as do also a small percentage of the very narrowest forms with a long free flagellum.

It was thought at one time that there might exist some connection between the virulence of the strain of trypanosome and the percentage of posterior nuclear forms. This has turned out, however, not to be the case. For in the T strain, where the posterior nuclear forms occur in such large numbers, the rats have lived from 17–29 days after inoculation, while in the G strain, where the posterior nuclear forms are very few or wanting, the rats have lived from 12–23 days, with the exception of one rat, the first inoculated from case G, which lived 38 days. With the exception of this last rat it is seen that the G strain has proved slightly more virulent than the T strain. The rats inoculated with the intermediate strain E have lived, however, 28–37 days, so that it is impossible to make any predictions of the virulence for rats of any particular strain of this trypanosome based on the percentage of posterior nuclear forms present in the rat. The results have been further controlled by the examination of two separate strains from G, one of which had been already started in rats by Dr. Newham, and had been maintained in rats and guinea-pigs for four months. The strains resemble one another in all essential respects. It is important to note the occasional absence of posterior nuclear forms in the rats infected with the G trypanosome. Unless repeated examinations of the rats are made it might be erroneously concluded that such did not occur. Though they may be absent on one day they are present on another.

The main points of the present note are these:—Firstly, in the subinoculated rats the posterior nuclear forms may vary very much in numbers in the various strains used: secondly, the number of posterior nuclear forms in the rats offers no criterion of the virulence of this particular strain as regards the rats themselves. A reference to Dr. Daniels's paper, however, will show that G, from whom the trypanosomes with the lowest percentage of posterior nuclear forms in the rats were obtained, appears to be infected with a trypanosome of lower virulence as regards man, while T and E, from whom the trypanosome strains with the higher percentages were derived, were certainly infected with trypanosomes of greater virulence. From the limited number of experiments and cases under observation, if any conclusion can be drawn it is this: if the trypanosome when inoculated into the rat shows many posterior nuclear forms, then that particular trypanosome is likely to prove very virulent for the individual from whom the strain of trypanosomes is derived.

III.—DEPARTMENT OF HELMINTHOLOGY.

By R. T. LEIPER, D.Sc., M.B., &c.

Gordius aquaticus as a Parasite in Man.

[F. Zschokke.—Proceedings of the Helminthological Society of Washington, April 1912.]

The adults of various species of *Gordiacea* have been found as occasional parasites of man, although they normally occur as free-living forms after they have emerged from the insect host in which they complete their larval development. The infection of man probably takes place through ingestion, in drinking water, of worms which have already completed their larval growth.

Dr. F. Zschokke adds another and apparently well authenticated case to the few already reported. The worm was about 17 cms. long, and was passed per anum by a Swiss boy of about 2½ years of age. The patient is said to have thereafter recovered from symptoms of a nervous character. The other species that have been recorded from man are *Gordius villote*, *Paragordius varius*, *Paragordius tricuspidatus*, *Parachordodes tolosanus*, *Parachordodes pustulosus*, and *Parachordodes violaceus*. R. T. L.

Fasciola hepatica as a Parasite in Man.

[Dr. Garrison.—Proceedings of the Helminthological Society of Washington, April 1912.]

Garrison reports having found an egg of a *Fasciola*, apparently *F. hepatica*, in a slide of human faeces, and notes that Ashworth has previously seen this egg in human faeces in Porto Rico. Commenting upon the demonstration, Stiles added that he had in his possession an adult specimen of this worm from a man in Porto Rico, and that cases had been reported from that island of death from asphyxiation as a result of living *Fasciola* attaching themselves to the throat during the consumption of raw liver. The prompt production of emetemesis would be likely to bring away the worms and so save the patient. The number of cases of *Fasciola* in man has now reached 28; but it is evident that the appearance of eggs in human faeces under circumstances such as those just related does not necessarily indicate that the eggs are derived from parasites normally inhabiting the liver of man.

R. T. L.

Symptoms apparently associated with *Tænia saginata*.

[Dr. Ransom.—Proceedings of the Helminthological Society of Washington, April 5, 1912.]

In a woman of 54 years of age who constantly passed portions of tænia, treatment with oleoresin of male fern brought about immediate and complete relief from the following symptoms:—Itching all over body, skin eruptions, cold feet, twitching of lower limbs, nausea, anorexia, flatus, occasional dizziness, and excessive leucorrhœa. R. T. L.

UNUSUAL SITUATION OF *ASCARIS LUMBRICOIDES*.

[DR. GARRISON.—Proceedings of the Helminthological Society of Washington, April 5, 1912.]

THE large round worm has not infrequently been found in the bile-ducts of the liver, in the appendix, and in the œsophagus and trachea. Dr. Kerr, Assistant Surgeon U.S. Navy, has recently brought under notice a specimen of *Ascaris* in the duct of Wirsung, extending across the pancreas from the intestinal termination of the duct to near the periphery. R. T. L.

A SECOND SPECIES OF *NECATOR*.

[LOOSS, A.—Records of the School of Medicine, Cairo.]

HITHERTO the genus *Necator* has comprised a single species, *N. americanus*, which is found as a parasite only of man and the gorilla. Looss has recently noticed in material received from Mr. Shipley that the *Necator* of the chimpanzee is uniformly smaller. There are remarkably small inner ventral teeth, the cutting plates at the edge of the mouth having rounded free edges; the lateral lobes of the bursa are broader than long, and the rays are comparatively thick and plump. This form has been named *Necator africanus*, but Stiles states ('Science,' p. 556, 1912) that this name is preoccupied by Harrison, 1910, as a synonym of *Necator americanus*. In an abstract of Prof. Looss's monograph in the 'Journal of the Royal Army Medical Corps' for July 1912, Cummins gives as a new term *Necator exilidens*; but it is not clear from the context if this is to be quoted in the literature as *N. exilidens*, Cummins, or *N. exilidens*, Looss. R. T. L.

REGENERATION OF LIVER AFTER *ECHINOCOCCUS*.

[DR. COBB.—Science, May 10, 1912.]

IN a discussion on a paper dealing with Cysticerci, Dr. Cobb draws attention to a remarkable case of hydatid in man in Australia. Owing to hydatid infestation a patient had a considerable part of the liver removed. The subsequent development of another cyst necessitated a second operation. The new cyst was found to be in another part of the liver, and it was observed that "there had been a practically complete regeneration of the excised portion of the liver." R. T. L.

THE TRANSMISSION OF ONCHOCERCIASIS.

[J. B. CLELAND.—Australian Medical Gazette, vol. xxxi., 1912.]

THE etiology of Onchocerciasis is of especial interest, not only on account of the occurrence of a species of *Onchocerca* [*F. volvolus*] in man, but also because the presence of large numbers of inflammatory masses due to these parasites in the Australian meat imported into England has recently led to active measures on the part of the home authorities in the interests of the consumer. Allied species present themselves in the camel and in the horse, in the latter leading to a considerable depreciation in working efficiency.

Dr. Cleland, who has devoted much attention to the subject, has recently had the unique opportunity of studying the etiological problem in the Island of Milson, which he had definitely shown to be an endemic area. The experimental animals were bred on the island, which is separated from the mainland by a stretch of water a quarter of a mile wide. No leeches or ticks have been found on the island, and Dr. Cleland abandons the hypothesis previously put forward that the life-history resembles that of the guinea-worm. He reviews as possible intermediaries, *Hæmatopinus vituli*, Mallophaga, *Stomoxys calcitrans*, Tabanids, Mosquitoes, and *Culicoides molestus*. Without any experimental data he is led to conclude that *Stomoxys calcitrans* is the most probable carrier. R. T. L.

On the Systematic Position of *Filaria perstans*.

[Railliet, Henry, and Langeron.—Le genre *Acanthocheilonema* et les Filaires péritonéales des Carnivores. Bull. Soc. Path. Exotique, vol. v. p. 392, 1912.]

M. Langeron has recently found in Tunis a dog infected with apparently the same species as that described by Cobbold under the name *Acanthocheilonema dracunculoides*. Two years ago the present writer had occasion to re-examine Cobbold's original of this species, preserved in the Hunterian Museum, and reported to the Zoological Society that the generic name was based upon caudal not oral appendages; whilst the alleged similarity to the guinea-worm, as indicated by the specific name, viz., the supposed absence of a vulva, was likewise arrived at from an inversion of the specimen. A male specimen occurred in the original material but was overlooked by Cobbold, who at that time supposed the guinea-worm to produce young parthogenetically. Attention was also drawn to the fact that the *Filaria perstans* of man exhibited the chief generic characters of *Acanthocheilonema*. The authors of the paper under review appear to have arrived independently at identical conclusions; they include, however, in the genus the two additional species, *Filaria recondita* and *Filaria grassi*.

R. T. L.

On *Filaria loa*.

[Verdun, P., and Bruyant, L.—Quelques documents pour servir à l'étude de la Filaire loa. Bull. Soc. Path. Exotique, vol. v. p. 506, 1912.]

From the study of a case of *Filaria loa*, in which the period of exposure to infection can be accurately determined as one year and fifteen days, the authors give the following conclusions:—From the history the period of incubation may be taken at a minimum of 5 months and a maximum of 17 months, which corresponds fairly well with the case reported by Ward. The worm when removed was found to be an adult male; the extraction occurred 22 months after the first symptoms of the appearance of a worm under the skin, and it is estimated that the worm afterwards removed from the eye was therefore about 3 years old. A high degree of eosinophilia persisted after the operation, indicating, possibly, the presence of other specimens of the *Filaria* or additional helminthic infection. R. T. L.

LACK OF PERIODICITY IN THE SHEATHED *FILARIA* EMBRYOS IN WEST AFRICA.

[THIROUX, A.—Les Filaires embryonnaires du sang des indigènes de l'Afrique Occidentale Française. Bull. Soc. Path. Exotique, vol. v. p. 438, 1912.]

IN an earlier number of this Journal reference was made to the results of Connal and others which showed that in Africa the periodicity of the embryos of *Filaria bancrofti* in some cases failed to conform with the classical description. M. Thiroux now contributes the results of a very exhaustive study of this question among the military forces in the French colonies of West Africa. Over 3000 men were examined. In 44 per cent. of the cases the sheathed *Filaria* was observed equally by day and night; in 42 per cent. it occurred by night only; whilst in 13 per cent. the embryos which were not observed by direct examination of the blood by night were recovered from blood during the day by means of the centrifuge. The embryos of *Filaria diurna* appear to retain more constantly their marked periodicity. In some cases a tendency to nocturnal periodicity was noticeable in *F. perstans*. R. T. L.

A NOTE FROM DR. MASSEY ON "EARLY SYMPTOMS OF ONCHOCERCIASIS."

A RECENT letter from Dr. A. Yale Massey, Lusambo, Belgian Congo, contains the following paragraphs, which appear to contain some new observations:—

"I am now sending you some *Filaria* from Calabar (?) Swellings. A native came to me with fever and painful swellings under the skin. They were situated on the sides of his chest, on the back, and on his legs. The largest were the size of a chestnut (horse). There was one on each side of the chest in the mid-axillary line, a little below the level of the nipple. These were the largest. They were tender to the touch, but the skin was not inflamed: the cyst moved slightly under the skin. After 10 days the fever was gone, and the small tumours remained, but not painful. It was then that I removed the one from the right chest, and on opening it I found it was a cyst containing a slightly milky fluid and knots of thread-like worms: the juice revealed filarial embryos. I am sending some knots of the *Filaria*, and some smears of the fluid, one stained."

[The specimens proved on microscopical examination to be portions of the female *Onchocerca volvulus*. The smears showed the large non-sheathed embryos typical of this form. So far as I know, these *Onchocerca* tumours have been regarded as painless, and the possibility of confusion with the Calabar swelling produced by *Filaria loa* has not hitherto been noted.—R. T. L.]

MR. LLOYD RECORDS A "NEW NEMATODE PARASITE OF *GLOSSINA MORSITANS*."

MR. L. LLOYD, Entomologist to the Luangwa Sleeping Sickness Commission, has written from Mpika (3rd March, 1912) regarding a Nematode which he has found parasitic in the common Tsetse-fly, *Glossina morsitans*. Mr. Lloyd proposes to join me later in a publication on the structural details and the systematic position of the worm. In the meantime the following extracts from his letters may prove of interest to other workers in Tropical Africa :—

"Four specimens have been met with. The first was in a tube in which several flies had died. When first seen the worm was free in the tube, dead and partly disintegrated. It was worthless as a specimen, nor could I be certain at the time that it had emerged from one of the flies, as my fly boys often place other things besides Tsetses in the collecting-tubes. The other specimens were found in flies that were being dissected. These were 'wild flies' which had been fed on a guinea-pig infected with Trypanosomiasis, and afterwards on monkeys, the flies being dissected as they died. The specimen that is mounted was found in a fly that had been in captivity for ten days. This fly was a female. The remaining two were found in a male fly that had been in captivity for nineteen days. It is therefore fairly certain that the parasites obtained access to the flies in nature. In each instance they were found closely coiled up in the body-cavity. Though the gut, proboscis, and salivary glands of the flies were examined, nothing was seen that could give any hint as to the history of the worms. In physiological salt solution they remained coiled, but when placed in water they uncoiled and moved about slowly. Placed again in salt solution, they coiled up once more. They were fixed in hot 70 per cent. alcohol, the unmounted specimens being forwarded in spirit of this strength. The unmounted specimens are both from the same fly."

He goes on to say :—"Should the worm prove, as I believe it to be, merely an insectan parasite, it will still be of great interest, as throwing some possible light on the question of the food of the Tsetse-fly. I take it that there are only two ways in which the parasite could obtain access to the fly—either from an animal upon which the fly was feeding or from water. It is a disputed point whether *Glossina* takes up water. On Lake Tanganyika, Dr. Kinghorn observed *G. palpalis* settling on the edges of puddles, while I have seen the same thing occurring with *G. morsitans* here on the Nyamadzi. When placed in a tube with a moist sponge or piece of blotting-paper, *G. morsitans* will settle on the moist surface and thrust in the proboscis; but I have never been able, by supplying coloured fluids, to prove that water was actually absorbed by the insects.

"The worm has been found in two flies only out of some three hundred wild flies that have been dissected here during the period of September to March. Both instances have occurred during the last two weeks, and the one seen in the tube was about a week previously to that. The rainy season is now drawing to a close, and for the last two months the low-lying plains and the hollows between the hills have been in a more or less swampy condition. The locality is a very dry one, practically no rain falling between April and November, while the swamps persist only for a few months of the year. Nawalia, where our laboratory is situated, is on the Nyamadzi River, a tributary of the Luangwa, and is at an altitude of about 2000 feet."

In a postscript he adds :—"Since writing the above I have found a fourth specimen of the Nematode to-day (4/3/12). It was in a male fly of the same series. The fly was very dry when found dead this morning, and the parasite dead. The blood of the animals upon which the flies were fed had been examined daily throughout the experiment."

[The specimens are all immature adult examples of a species of *Mermis*, and resemble somewhat closely that found by Professor Minchin a few years ago in *Glossina palpalis* in Uganda.—R. T. L.]

TRICHINOSIS.

[ROMANOVITCH in Annales de l'Institut Pasteur, vol. xxvi. no. 5, 1912.]

FROM new researches on Trichinosis, Romanovitch arrives at the following conclusions :—

(*a*) The female of the Trichina penetrates into the thickness of the intestinal wall. She stops usually in the chorion of the mucous membrane, but does not go any further than the "muscularis mucosæ," and, contrary to the assertions of Cerfontaine, does not reach the mesentery glands.

The female deposits the larvæ either in the lymphatic vessels or in their neighbourhood. The larvæ follow the lymphatic channel to reach the circulatory system. It would be useful, therefore, where suspected meat has been consumed, to make a daily examination of the blood. In this way the infection could be detected from the very commencement.

(*b*) The epithelial cells of Lieberkühn's glands often show numerous karyokinetic figures, not only in the immediate neighbourhood of the lymphatic vessels invaded by Trichina, but also at some distance from them.

(*c*) The larvæ may reach the peritoneal, pleural, and pericardial cavities, but dies very rapidly.

(*d*) It must be accepted now as definitely demonstrated that the Trichina larva actually penetrates into the muscle fibre itself, because it finds there, better than anywhere else, the nutriment necessary for its development.

(*e*) In passing through the intestinal mucosa, the Trichina, soiled with microbes, scatters them on its way. As the bacteriological study of the blood of infected human beings and animals shows, the dominant character of a Trichina infection is polymicrobial.

It is difficult to deny that the fever, abscesses, and fatal septicæmia which sometimes occur in man are due to bacteria inoculated by the Trichinæ.

(*f*) Animal serum (rats, guinea-pigs) infected by Trichina acquires toxic properties, which show nine days after the consumption of trichinosed meat. These sera are toxic for the guinea-pig as well as for the rat, that is to say, that they are at the same time iso- and "hetero"-toxic.

The toxicity of the serum of an infected guinea-pig is in proportion to the intensity of the larval infection. The sensitiveness of animals to these toxic substances is variable and subject to individual variations. The animals which survive after the injection of toxic serum are at the end of a few days extremely thin. The urine of highly infected animals may also prove toxic.

(*g*) Research after specific antibodies in serum of trichinosed animals has not given satisfactory results, neither by the precipitin method nor by fixation of complement. In these experiments aqueous extract of muscle full of larvae was used as antigen.

(*h*) Cases of spontaneous re-infection confirm the experiments of Rupprecht, Askanazy, and others, which demonstrated the impossibility of immunising animals from fresh infection.

(*i*) There is no preventive or abortive treatment for trichinosis. Sometimes the evolution of this helminthiasis can be retarded by emetic injection. Ehrlich's 606 does not seem to exercise any action on the larvæ.

V. A. I.
R. T. L.

The Liver-fluke of the Indian Pariah Dog.

[J. W. W. Stephens.—Annals of Tropical Medicine and Parasitology, vol. vi. 1. B, 1912.]

Gaiger has recently shown that the liver-fluke, which has been known for some years under the name *Opisthorchis noverca,* is extremely common in dogs in the North-West Provinces, occurring in 19 out of 50 consecutive post-mortems at the Punjaub Veterinary College. In 1876 McConnell described from the bile-ducts of two Mahommedans some small flukes, and these have been regarded as identical with those from the Indian dog until the present year. In a paper published as No. 103 in the series "Studies from the Zoological Laboratory, Univ. of Nebraska," Professor Barker splits off a number of species from the genus *Opisthorchis* and incorporates McConnell's specimens in the new group, to which he gives the name *Amphimerus.* Thus he regards the *Opisthorchis noverca* of man not only as distinct specifically from that of the dog, which he renames *caninus*, but actually separates them into different genera. In the present paper Stephens describes very carefully and fully the anatomy of the dog-fluke, and likewise concludes that it does not correspond to *Opisthorchis noverca* of man. Adopting Barker's specific name *caninus*, he proceeds likewise to separate the species from the original genus, but ignoring Barker's genus *Amphimerus* places this form in another new genus which he names *Paropisthorchis.* The existence of a process or neck supporting the ventral sucker is regarded as of generic value. From an examination of material in the Helminthological Department, the present writer does not feel certain that the specimens collected from man by McConnell are rightly to be regarded as distinct from those found in dogs. It is true that McConnell did not see or figure the pedicle or neck of the ventral sucker; but this structure is not at all obvious, especially in mounted and cleared specimens, nor is it *constant* as such. In some of the specimens recently examined the whole pedicle was retracted, and the ventral sucker actually lay in a hollowing of the ventral surface; while the genital pore in these cases could be seen lying in front of the ventral sucker as described by McConnell. Certain of these specimens from the dog also showed the striking irregular and collapsed appearance of the testicles figured by McConnell. It is doubtful if the pedicle could be regarded as a feature of higher than specific rank in the *Opisthorchiinæ*, even were it absolutely indisputable that McConnell's specimens did not possess this peculiarity and were not merely retracted forms. R. T. L.

SOME HELMINTHOLOGICAL OBSERVATIONS IN NYASALAND.

By HUGH S. STANNUS, M.D. (Lond.), D.T.M. & H. (Cantab.),
MEDICAL OFFICER, NYASALAND.

DURING the early part of 1912, one or two inmates of the Central Native Prison, Zomba, came under my notice exhibiting a marked grade of anæmia. Infection with ankylostomiasis was suspected, and this diagnosis was confirmed by finding ova in the stools.

Having seen cases of ankylostomiasis from time to time among natives in other districts, I thought it would be interesting to make an examination of the stools of a large number of individuals with a view to determining the incidence of ankylostome infections. I therefore submitted to microscopical examination the fæces of all the prisoners in the Central Gaol, numbering 230 persons. The results are embodied in Table I.

Investigations were then extended to the Native Troops stationed in Zomba and a number of Reservists who came in for a month's training, Tables II. and III.

The figures obtained for women and children at the Camp are given in Tables IV. and V., and for the Civil Hospital in Table VI.

In the first three tables, the first column gives the districts of the Protectorate from north to south, with the number of men from each in the second; the number of cases of ankylostome infection is placed in the third column, while in successive columns are put the figures relating to other helminths whose ova or larvæ were found during routine examination. Mention is also made of the Protozoon *Balantidium coli* and of an Acarid.

It will be seen that infection is found among natives from practically all districts of the Protectorate; the number of infected is roughly proportional to the total number of men from any particular district (Table I.). The figures for the Troops bear this out. It might be argued that infection was gained in the Central Prison, but other facts go to show that the disease is endemic elsewhere in the Colony. I have seen cases in Fort Johnston, Blantyre, and North Nyasa Districts, and prisoners on admission were found to be already infected. The figures relating to Reservists may be taken to point to the same conclusion. As far as Zomba is concerned, the disease is found among the village population (Table VI., Civil Hospital) just as at the Prison and Camp.

I think, therefore, the figures arrived at may be taken as representative probably of the conditions all over the Protectorate.

Dealing with the actual figures, I have shown that 26 per cent. of the inmates of the Central Prison harbour ankylostomes; for Native Troops the figure is 15·7 per cent., and for Reservists 18·6 per cent. Among the few women examined at the Camp, 38 per cent. were infected, but of 12 children under two years of age none were affected. The figures for the general population included both sexes and all ages over two years; the percentage of infected was 19·0, among whom were a number of children between two and five years of age. Excluding the few children under two years of age, of 493 persons whose stools were examined 22·2 per cent. were found to be infected.

Bass has shown that by the method of diagnosis adopted (examination of fæces for ova) some 20 per cent. of cases are missed.

Making such a correction one arrives at the figure 42 as representing the probable true percentage of infected. Turner found among natives recruited from Nyasaland, Mozambique, &c., for the Transvaal Mines. 50 per cent. of infected.

The important question of the degree of infection may be here considered. It is necessary to distinguish between those who are simply infected ("carriers") and those who are suffering from the effects of the infection ("diseased")—in other words, those infected with ankylostomes and those suffering from ankylostomiasis.

Bass estimates that one egg per microscopical slide roughly indicates an infection with less than 10 worms, while Nicol, in India, has given it as his opinion that to produce effects on the host there must be 500 worms present for six months.

In a large majority of my own cases there have been 1–2 eggs per slide, with a maximum of 7. This would point to a low infection, and such is confirmed by the results of treatment and expulsion of worms and post-mortem findings. Among a number of cases treated, the greatest number of worms expelled was 14, more usually some 3–10. I have so far only had the opportunity of making post-mortem examinations on the bodies of two natives who, during life, showed ankylostome ova in the fæces: in one 25 worms were found, in the other 9.

Of the 100 odd cases of infection, 15 complained of symptoms or showed signs of the infection. Ten showed a recognisable degree of anæmia, in two of these the anæmia was marked: three had hæmic murmurs, three complained of abdominal pain, constipation, &c.; in two there was marked stomatitis. It is, however, very probable that slighter grades of anæmia, loss of weight, joint pains, ineptitude for work, &c., symptoms of the disease, have passed unnoticed.

Dr. Leiper has very kindly examined a number of worms from these cases, and reports that both *Ankylostoma duodenale* and *Necator americanus* occur in the material submitted.

Mention may here be made of other parasites met with.

Reference to the Tables shows a proportionately small infection with other worms. Among these the ova of *Tricocephalus trichiuris*, *Tænia* sp., *Dibothriocephalus* sp., and *Schistosomum hæmatobium* were found, and the larvæ of *Strongyloides intestinalis*.

Balantidium coli was found in three cases, and an Acarid on two occasions.

It is interesting to note that tapeworm eggs were only found among the Reservists, and *Balantidium coli* only among Prisoners.

A rather remarkable point, however, is the absence of any case of infection with *Ascaris lumbricoides*. Bilharzia ova were found in 3·8 per cent. of the total number of natives examined. In all cases the ova were of the lateral-spined variety. In none of these were symptoms referable to the infection present, and none admitted ever having had symptoms of urinary bilharziosis.

TABLE I.—*Prisoners. Central Prison.*

District.	No. of Men.	Ankylostome.	Schistosome.	Strongyloides.	Tricocephalus.	Tænia.	Bothriocephalus.	Ascaris.	Balantidium.	Acarid.	Remarks.
N. Nyasa	5	1	..	..	..	..	..	..	..	..	
W. Nyasa	2	..	..	..	..	..	..	..	..	..	
Momberas	22	6	..	..	..	..	..	..	..	..	
Marimba	12	7	3	..	..	..	..	..	..	..	
C. Angoniland ..	31	8	1	..	..	..	..	..	..	..	
S. Nyasa	19	3	..	..	..	..	..	..	..	..	
Upper Shiré	46	6	3	..	..	..	..	..	1	..	
Zomba	12	4	..	..	1	..	..	..	..	..	
Blantyre........	50	14	3	1	..	..	..	..	2	..	
Mlanje	24	6	1	..	..	..	..	..	..	..	
W. Shiré	..	..		..	..	..	..	..	..	..	
Ruo	4	1	1	..	..	..	..	..	..	..	
Lower Shiré	3	1	..	..	..	..	..	..	..	..	
	230	57	12	1	1	0	0	0	3	0	Totals.

TABLE II.—*Native Troops.*

W. Nyasa	2	1	..	..	..	..	..	..	..	..	5 multiple infections: 1 Schistosome and Tricocephalus; 1 Ankylostome and Tricocephalus: 1 Ankylostome and Schistosome; 1 Schistosome and Strongyloides and Tricocephalus.
C. Angoniland ..	2	..	..	..	..	..	..	..	..	..	
S. Nyasa	46	8	2	1	2	..	..	.	..	1	
Upper Shiré	19	3	1	3	4	..	..	..	..	..	
Zomba	16	5	..	2	1	..	..	..	..	..	
Blantyre........	1	..	..	..	..	..	..	..	..	..	
	86	17	3	6	7	0	0	0	0	1	Totals.

TABLE III.—*Reservists.*

District.	No. of Men.	Ankylostome.	Schistosome.	Strongyloides.	Tricocephalus.	Taenia.	Bothriocephalus.	Ascaris.	Balantidium.	Acarid.	Remarks.
N. Nyasa	1	..	..	..	..	..	..	..	..	..	10 multiple infections, always Ankylostome with some other.
W. Nyasa	9	2	..	..	1	..	..	..	..	..	
Marimba	1	..	..	..	..	..	..	..	..	..	
C. Angoniland ..	4	..	..	..	..	..	..	..	..	..	
S. Nyasa	1	1	..	..	..	..	..	..	..	..	
Upper Shire	26	..	..	..	2	..	..	..	..	..	
Zomba	42	10	2	6	..	4	1	..	..	..	
Blantyre...... ..	20	3	..	1	..	..	1	..	..	..	
Mlanje	10	2	..	..	..	..	..	..	..	..	
	114	18	2	7	3	4	2	0	0	0	Totals.

TABLE IV.—*Women.*

Camp	21	8	.	2	2	..	..	..	..	..	2 double infections: 1 Ankylostome and Strongyloides; 1 Strongyloides and Tricocephalus.

TABLE V.—*Children under 2 Years.*

Camp	12	..	..	..	..	..	..	..	..	..	No helminths.

TABLE VI.—*General Population.*

Zomba	52	8	2	..	..	..	..	..	..	1	Of the 8 infected with Ankyl. 4 were children between ages of 2 and 12 years.
Protectorate ..	515	109	19	16	13	4	2	0	3	2	Grand Totals.
Or excluding Infants......	503	109	19	16	13	2	2	0	3	2	
Per cent. Infected	..	22·1	3·8	3·2	2·6	..	..	..	..	..	

Besides the cases of infection with *Balantidium coli* mentioned above, I have seen others in which the organism swarmed, and these all without any symptoms.

The acarid was not identified; probably it was derived from food and had survived its passage through the gastro-intestinal canal.

It is unnecessary here to enter into the questions of methods of infection with ankylostomes, they are the same as in other countries where similar conditions prevail.

Treatment of individual cases can be carried out, but preventive treatment on a large scale is still a matter for the future.

IV.—PATHOLOGY.

By H. B. NEWHAM, M.R.C.S., D.P.H., &c.

Abstract from the 'Philippine Journal of Science,' Vol. vi. No. 6:
Section B. Medical Sciences, December 1911.

The Occurrence in the Philippines of associated Spirochætæ and Fusiform Bacilli in Ulcers of the Throat (Vincent's Angina), of the Mouth, and of the Skin, and in Lesions of the Lungs (Bronchial Spirochætosis).

By WESTON P. CHAMBERLAIN

(Major, Medical Corps, United States Army, President of the United States Army Board for the Study of Tropical Diseases as they exist in the Philippine Islands).

In an introduction to this careful and interesting paper, which is well illustrated, the author mentions that, in the report of the Board of March 1910, brief reference was made to the finding of several cases of so-called Vincent's Angina in the Philippines. Since then a look-out had been kept for the presence of Vincent's symbiotic organisms in all throat-lesions, and a large number of cases had been detected. Plaut (1894) and Vincent (1896) had called attention to a fusiform bacillus commonly associated with a spirillum in ulcerative lesions of the throat, pharynx, and mouth. Many writers have since confirmed these observations, and the two organisms have been found in various other kinds of lesions. Weaver and Tunnicliff have demonstrated the organism in noma, and others have found them in ulcerative stomatitis, diphtheria, carious teeth, hospital gangrene, appendicitis, brain-abscess, fœtid bronchitis, pyorrhœa alveolaris, and syphilitic lesions. Vincent, Smith and Peil, Bruce, and the author have found similar forms in skin-ulcerations. Recently Peters, in Cincinnati, reported a case of fatal lobar pneumonia, a case of fœtid bronchitis, and a case of septic infection of the hand—in all three of which the exudate showed large numbers of spirochætæ and fusiform bacilli. The hand-infection had followed injury by the teeth of another person. Hultgen recorded a case of hand-infection in a patient with nail-biting habit, whose teeth harboured the same organisms.

Bacillus fusiformis of Plaut and Vincent varies from 3 to 15 μ in length. The author has occasionally seen much larger ones. The bacillus tapers toward the ends, sometimes blunt, at others sharp. They may be straight or sharply curved, and are often in pairs end to end. Occasionally wavy forms are seen. They are non-motile. They stain readily, many showing irregularity in the intensity of the staining, giving them a characteristic banded appearance.

The spirochætæ are longer than the bacilli, ranging from 15 to 50 μ, the undulations being from 3 to 5. They are usually very slender, and in fresh preparations extremely active. They stain less intensely than do the fusiform bacilli and may present a beaded appearance. The author has failed to cultivate these organisms after several attempts. Weaver and Tunnicliff grew them in pure culture on slants of ascitic agar, and Peters on Dorset's egg medium—in both cases under anærobic conditions. Tunnicliff and some others regard bacillus and spirochæte as different forms of one organism. Varying numbers of staphylococci, streptococci, and different species of bacillus, including that of Klebs-Loeffler, are usually found with the symbiotic organisms of Vincent's angina in smears from oral and faucial ulcers. Abel has found the organisms in normal mouths, and Gross in apparently healthy tonsils.

It has been suggested that to make a diagnosis of Vincent's angina the organisms should be present in preponderating numbers as compared with other bacteria. This distinction, though unsatisfactory, may be of some clinical use, and is, to a certain extent, adhered to in this report.

During the last two years 106 patients with throat and mouth lesions were examined in Manila. In smears from 34 (32 per cent.) the fusiform bacilli and spirochætæ were present in *preponderating numbers*. In 22 (21 per cent.) a few of these symbiotic organisms were found, so that in 53 per cent. of the total 106 cases they were present to a greater or less extent. The pathologic conditions with which they were associated were :—ulcers of tonsil, cause uncertain : ulcers of tonsil, gums, soft palate, and tongue, all probably syphilitic ; ulcers of gums (ulcerative stomatitis) ; membranes on tonsil, cause uncertain ; syphilitic mucous patches ; acute follicular tonsilitis ; carcinoma of soft palate.

The number of positive findings were highest in ulcers of the gums and tonsils. Twenty-seven of the lesions tabulated were apparently syphilitic, 2 primary, 2 tertiary, and the remainder secondary ; and in these the organisms were found in preponderating numbers in 10 and in small numbers in 1. In not all of these presumably specific cases was the diagnosis absolutely unassailable, but in some of those in which the fusiform bacilli and spirochætæ were demonstrated in large numbers the diagnosis of syphilis was confirmed by the finding of *Treponema pallidum*, by the presence of a positive Wasserman reaction, or by the existence of unmistakable physical signs outside the mouth.

In four cases where the Klebs-Loeffler bacilli were found by cultural methods, fusiform bacilli and spirochætæ were absent. In many of the cases showing these organisms in large numbers the local appearances were strongly suggestive of diphtheritic infection : cultures on blood serum, however, were negative, as far as the diphtheria bacillus was concerned. Nearly all the positive cases were among American soldiers, only one case being found among the Filipinos. The clinical appearances and symptoms were so variable that diagnosis based on them would have been valueless. The frequent presence of spirochætæ and fusiform bacilli in the mouth might be due to carious teeth : an examination of material from such teeth in ten individuals revealed in three cases an occasional spirochæte, but in no instance were fusiform bacilli seen. A number of smears from apparently normal tonsils showed in a few cases scanty spirochætæ and very rarely a small fusiform bacillus.

In Skin-lesions in the Tropics.—Castellani and Chalmers are quoted that "Vincent believes ulcus tropicum to be due to the association of spirochætes and fusiform bacilli so frequently found in such ulcers. Vincent's observations have been confirmed by Smith and Peil in Sierra Leone, Patton in Aden, and many other observers in various parts of the tropics."

In 1907 Shattock, in the Philippines, reported that of 34 ulcers of the skin five contained spirochætæ in the exudate. Some resembled *Spirochæta refringens*, others appeared intermediate between the latter and *Treponema pallidum*. Owing to their absence in sections, he concluded they were of no etiological significance. In his photo-micrographs are some large, not very clearly defined bacilli resembling *B. fusiformis*, though he makes no mention of them.

Howard, in Nyasaland, reports spirochætæ generally with *B. fusiformis* to be common in various types of ulceration; and in sections the spirochætæ can be seen spreading into the healthy tissue in advance of other micro-organisms.

W. J. Bruce, in Jan. 1911, reported, as "Zambesi ulcer," punched-out ulcers below the knee, healing in one or two weeks and without any constitutional symptoms. Smears from these ulcers always showed large numbers of a spirillum and a large fusiform bacillus.

Shortly before Bruce's report, in an ulcerated area on the foot of a Filipino in which no *Treponema pertenue* could be detected numerous spirochætæ and large fusiform bacilli were found. During two years smears from a large number of skin-ulcers have been examined; but these organisms were not found on any other occasion. Strong and others, in Manila, have not encountered them in lesions of the skin.

Recently, Mueller has found several such cases near Iloilo.

Bronchial Spirochætosis with an occasional Fusiform Bacillus.—Bronchial spirochætosis was first described by Castellani in 1906. Since then, many cases, acute and chronic, have been reported by observers in various tropical countries. Most of these diagnoses appear to have been made from sputum-examinations alone.

In 1909, Phalen and Kilbourne, in recording a case in a Filipino, called attention to the fact that the causative relation of the spirochætæ to the disease was not proven. A few large bacilli were present; on re-examining recently smears from this case, a few perfectly typical fusiform bacilli were found with the characteristic barred appearance when stained.

Recently sputa of two Filipinos suffering from typhoid fever with cough were found to contain large numbers of actively motile spirochætæ; in both cases similar in motility, shape, and size, averaging 15 microns in length. They were thin, delicate, and resembled the third class described by Castellani, except that none having one blunt point was seen. In one of the patients were found a few fusiform bacilli, like those seen in symbiosis with spirochætæ in the so-called Vincent's angina. In history, symptoms, and character of sputum these two cases were almost identical, except that one contained fusiform bacilli and the other did not. The spirochætæ persisted in the sputum far into convalescence.

Specimens of sputum from 5 natives and 27 Americans having cough with expectoration were examined. In only one were spirochætæ found. These were a few thick spiral organisms such as are common around necks of the teeth.

Prevalence of Spirochætæ and Fusiform Bacilli in Lesions occurring in non-Tropical Countries.—These associated organisms would probably be frequently met with if the practice of examining smears from ulcers in those regions was more general.

Rodella found the two organisms present in one-third of 2000 cases of pseudo-membranous anginas which he examined. Beitzke in 5 out of

58 patients suspected of having diphtheria. Sublowitz in 6 out of 38 ulcers of the mouth. Arnold in 3 out of 5 cases of follicular tonsilitis. Rothwell, under the title "Bronchial Vincent's Angina," reports 4 cases in Missouri where sputum was swarming with fusiform bacilli and spirochætæ—there were asthma-like symptoms and bloody expectoration. The presence of these two organisms has been reported in the sputum of cases of fœtid bronchitis and pneumonia. There are so few reports, however, that no reliable estimate can be made of the real frequency of the condition in pulmonary cases in temperate regions.

The organisms may easily go undetected, for most sputa are examined microscopically for tubercle bacilli. The spiral organisms in pulmonary spirochætosis stain well with carbol fuchsin or Loeffler's methylene-blue. They are, however, not acid-fast, and do not stain with the sulphuric acid methylene-blue as ordinarily used in Gabbett's method for demonstrating tubercle bacilli. This is true also for the spirochætæ in Vincent's angina.

The occurrence of the organisms in infections of the skin in temperate regions has been reported but rarely, and no estimate can be formed of the frequency of such lesions.

Major Chamberlain concludes that, from the observations reported in this paper, it is probable that more than one-half of the throat and mouth ulcers encountered in the Philippines will show fusiform bacilli and spirochætæ, and in preponderating numbers in one-third of them. It must be remembered that, in all the cases examined, these particular parasites were specially looked for.

The associated organisms have been found in an ulcer of the skin and in two cases of pulmonary disease, but as yet there is no evidence to show that in the Philippines they are common in these situations.

One caution needs to be given. The finding of fusiform bacilli and spirochætæ in a throat-lesion does not justify resting content with a diagnosis of "Vincent's Angina." Experience shows that syphilis, diphtheria, carcinoma (and very probably other etiological factors) must be carefully ruled out. It is still an open question whether the associated fusiform bacilli and spirochætæ are ever causative of the multiform lesions in which they are so often found to be present. H. M. H.

ABSTRACT FROM 'JOURNAL OF PATHOLOGY AND BACTERIOLOGY,' Vol. xvi.

AUTO-ERYTHROPHAGOCYTOSIS IN PROTOZOAL DISEASES.

BY ANDREW CONNAL, M.D., D.P.H., D.T.M. & H.

DR. CONNAL, in an introduction to this paper, notes that erythrophagocytosis has been described by Hektoen (1907), Davis (1907), Wright (1906), Macgregor (1909), Barratt (1905). Excepting Wright, all these observers describe the phenomenon *in vitro* only. Macgregor says: "Rabbits rendered immune to goat's blood furnish a serum that sensitises the red cells of the goat for phagocytosis; the spontaneous occurrence of such bodies (erythro-opsonins) in disease is, however, much rarer."

Hektoen observed the presence of erythro-opsonins as well as erythro-agglutinins in typhoid fever, pneumonia, erysipelas, cellulitis, and scarlet fever. Wright notes the occurrence of auto-erythrophagocytosis in a case of pneumococcal cellulitis of the neck and in one of pneumococcal cystitis. All these observers mention the co-existence of erythro-opsonin with erythro-agglutinin. Apparently also only the neutrophil polymorphonuclear leucocytes were phagocytic towards the red cells.

Kanthack, Durham, and Blandford first described auto-agglutination of the red-blood corpuscles in trypanosomiasis of the lower animals. Recently, Warrington Yorke (1911) has investigated the subject. None of these observers, however, mentions auto-erythrophagocytosis.

Dow (1908) gives details of a large series of blood-examinations in cerebro-spinal fever (a disease characterised by much wasting and anæmia in its later stages), but he never observed any signs of ingestion of the red cells by the leucocytes.

The object of this paper is to draw attention to the process of auto-erythrophagocytosis which has been found to occur in the peripheral blood in many cases of protozoal disease in West Africa. The scope of the inquiry has been limited to Southern Nigeria, Togoland, and the Gold Coast. The observations detailed are based on a careful scrutiny of the blood of man and of the lower mammals, both of those apparently in health and of those suffering from various protozoal and other infections. The human diseases investigated are trypanosomiasis, malaria, yaws, syphilis, filariasis, beri-beri, yellow fever, small-pox, leprosy, and ankylostomiasis. The animals, besides man, whose blood has been examined are cows, horses, sheep, goats, pigs, dogs, rats, and guinea-pigs. A differential leucocyte count was made in every case, the percentages being based on the enumeration of at least 500 white cells—a procedure adopted to ensure a careful survey of the individual leucocytes. Fresh blood-films were examined as often as was practicable; the stain used for the dried films was a simple modification of the eosin-azur mixture. The results of observations on over 800 different cases, involving over 1000 blood-examinations, form the basis of this paper. The percentage of macrophages, in the cases where these cells occurred, varied between 3.5 and 0 2, and as a rule it was less than one.

Cases of Trypanosomiasis.—Auto-erythrophagocytosis was first noticed in the blood of a woman in Lagos. Trypanosomes were fairly numerous in the peripheral blood during the first three or four days of her residence in the hospital—they diminished rapidly in number under treatment with potassium iodide and liquor arsenicalis. The species appeared to be *T. gambiense*. Erythrophagocytosis was most marked on first day of examination, decreasing steadily thereafter. Numerous fresh blood-films were examined on different days, but the actual phagocytosis in process was never seen, nor was auto-agglutination of the red cells observed. Various fallacies had to be excluded in searching for evidence of phagocytosis. (1) In a thick blood-smear, or in one that had not dried quickly, mononuclear leucocytes were seen with protoplasmic processes almost completely wrapped round red cells, the latter apparently having been fixed first, the leucocyte retaining its amœboid movement a little longer. (2) In a thick smear, super-imposition of red cells on leucocytes. The signs relied on for diagnosis were: (*a*) Nucleus and cytoplasm of the macrophage in the same focal plane as the contained red cell; (*b*) the presence of macrophages containing bodies in various stages from the normal shape and size of erythrocytes, but of rather paler colour, to a condition resembling merely vacuoles of different sizes. The nucleus and protoplasm of the latter stained equally well with those of the other varieties of leucocytes ; the vacuoles were not therefore simply degeneration-products of the erythrophage cytoplasm. It is believed that the phagocytic process occurred *in vivo.* The smears were made thin so as to dry instantaneously, and were always fixed and stained within a few minutes. The number of red-blood cells per c.mm. diminished while phagocytosis was active, and rose again when the process ceased. Two types at least of large mononuclear cells could be distinguished : (*a*) the ordinary large hyaline cell ; (*b*) the other was larger, irregular in outline, elongated, pointed or square, the nucleus occupying not more than half the cell and of a horseshoe or irregularly rounded shape. The cytoplasm was faintly granular and stained bluish-pink. The nucleus stained a rich purple. This second type is what is here termed a macrophage or erythrophage *when it is seen to contain a red cell.* It differs from a neutrophile myelocyte in the staining characters of its nucleus and cytoplasm. As a rule, two or three red cells were found ingested by the one phagocyte. Leucocytes containing red cells were, as a rule, larger than the others. The protoplasm of the ingesting cell was never seen bulged outwards by the contained erythrocyte, but there was often an indentation of the nucleus, where two red cells had been taken up, one on each side of the nucleus ; the latter frequently showed the appearance of hourglass contraction.

Possibly a few of the vacuolated mononuclear cells corresponded to the plasma cell or "irritation cell" of Türck, and possibly also some of them were vacuolated myelocytes.

Serum was obtained from the patient while the evidence of phagocytosis, although less than before, was still present. Blood was withdrawn in Wright's capsules and the serum allowed to separate out. It was centrifugalised to still further ensure the absence of cellular elements. Red and white corpuscles were then taken from five different sources : (1) Healthy European male ; (2) ditto female ; (3) case of advanced sleeping sickness in a child ; (4) an adult native infected with *Filaria perstans* ; (5) European with heavy and untreated infection with malignant malarial parasites. The corpuscles were washed with sodium citrate and with saline, and the top layer containing mixed white and red cells was used. Four sets of experiments were carried out with each person's cells :—

(1) A pipette with 1 part person's cells to 1 part patient's serum incubated at blood-heat for 15 minutes.
(2) A pipette with 1 part person's cells to 1 part patient's serum incubated for 30 minutes.
(3) A pipette with 1 part person's cells to 2 parts patient's serum incubated for 15 minutes.
(4) A pipette with 1 part person's cells to 2 parts patient's serum incubated for 30 minutes.

No evidence of phagocytosis was seen in any of the smears made from the contents of the pipettes, so that hæmopsonin was probably absent. Wright suggests that toxic damage may have been done to the red cells leading to auto-phagocytosis without the intervention of opsonin.

Observations were made on 20 other cases of trypanosomiasis, in all of whom, except one, the parasites had been demonstrated at one time or another, either in the peripheral blood or in the gland-juice. Erythrophages were seen in 14. Trypanosomes were present in the smear at the same time in only 3 of these. 3 cases in the 20 showed trypanosomes and no macrophages. Vacuolated cells, however, were found in the entire 20. The number of cases is not large enough to enable a connection to be established between the circumstances of the disease and the occurrence of macrophages. Treatment directed against the trypanosomes apparently puts an end to the phagocytic process.

Examination of Lower Animals.—Eighteen rats were examined: 5 harbouring *T. lewisi* showed no macrophages, although a few vacuolated mononuclear cells were seen in all the films. These 5 cases compared with 10 others in which no parasites were found showed only slight differences in the percentages of the various white cells. It is noted that *T. lewisi* is non-pathogenic. A rat inoculated with blood from a heavily infected cow did not develop any trypanosomes. At the time this rat exhibited the intracorpuscular stage of some parasite, the condition being suggestive of the fragmentation stage of spirochætes described by Balfour and Sambon. One macrophage was seen 6 days after inoculation, and on four subsequent occasions vacuolated mononuclear cells were noted.

A rat inoculated with blood from a heavily infected horse developed the trypanosomes in 3 weeks; both macrophages and vacuolated mononuclears occurred. The species of trypanosome in the last 2 cases is not known. It was not *T. dimorphon*.

A white rat inoculated with *T. rhodesiense* showed no macrophages. Two horses out of eight examined showed trypanosomes (species undetermined). Macrophages and vacuolated cells were found in one; neither in the other. The parasites were much the more numerous in the former; the latter had been treated with subcutaneous injections of serum obtained from a "salted" horse. 42 sheep were examined and one was found infected with numerous trypanosomes, apparently similar to the one found in the cows. Macrophages and vacuolated cells were found in the blood of this animal. In 66 films from pigs trypanosomes were found once. They were not numerous and their species was uncertain. Neither macrophages nor vacuolated mononuclears were seen.

Trypanosomiasis was found to be fairly common in *cows*, more especially in the large "humped" cattle brought to the coast from up country. 16 animals out of 56 examined showed trypanosomes, all apparently of the same species.

Macrophages were found in the blood of 4, and vacuolated cells in 7 cases. Smears were obtained from liver and spleen of 5 at time of slaughtering. In three cases macrophages were seen in smears from both organs. They had also been seen in the peripheral blood. In two cases, erythrophages were seen in both organs of one, and in the liver alone in the othar case, although they had not been observed in the blood of either. 2 guinea-pigs were inoculated from a horse with trypanosomiasis. The parasites developed in one case, not in the other. One macrophage was seen in smears taken four weeks later from each. Vacuolated cells were found in blood of both.

A bulldog was found naturally infected with trypanosomes. They were absent in 16 other dogs examined. The parasites were numerous and the animal much emaciated and vicious. No macrophages or vacuolated cells were found in the blood. The blood of 17 goats was examined, but no parasites were seen.

Animal Diseases other than Trypanosomiasis.—Erythrophages were observed, apart from trypanosomiasis, in the blood of 2 sheep, 2 cows, and 1 goat. A parasite resembling a nearly full-grown malarial form was seen in one of the sheep. No parasites were seen in the other. The two cows each gave a high eosinophile count. No blood-parasites were detected. The goat's blood showed a high polymorphonuclear percentage and parasites were apparently absent.

A hæmogregarine was seen in the blood of one rat which also harboured *T. lewisi.* There were a few vacuolated cells but no macrophages. Intro-corpuscular forms of some parasite were found in another rat. Erythrophages and vacuolated cells did not appear.

One pig was found to be heavily infected with piroplasma. There was no evidence of erythrophagocytosis.

Filarial embryos were very abundant in the blood of one dog. No macrophages or vacuolated cells were found.

Malarial Cases.—The erythrophagocytic cells in this series were similar to those found in the cases of trypanosomiasis, but, in addition, in a few instances, the typical large hyaline mononuclear cells were seen containing ingested red cells. No opsonic experiments were made with the malarial cases. Auto-agglutination of the red cells was observed in only two cases where macrophages were present. Usually only one red cell was found ingested by one leucocyte; in a few instances two or even three erythrocytes were seen in the one cell. In some cases blood-platelets were seen to have been ingested. Careful search was always made for a phagocytosed red corpuscle containing a malarial parasite; this was found in 2 cases.

Children æt. 1 year.—67 were examined; only 9 of these were brought for treatment for "fever." Malarial parasites occurred in 54. 12 cases of benign tertian infection, erythrophagocytosis occurring in two; 16 of quartan infection with macrophages in 6; malignant tertian 8, with ingestion of red cells in 8; benign and malignant together, 4 cases, phagocytosis being seen in 2; quartan and malignant tertian co-existed 10 times, macrophages being seen in 4. Benign and quartan parasites were found in 2 cases, macrophages occurring in both. All three species of parasite together in 2 cases, erythrophages being found in both.

Phagocytosis occurred in 21 cases, and in 57 vacuolated cells were observed.

Children æt. 2 years.—In 31 out of 35 parasites occurred. Five of the children were brought for treatment for illness referable to malaria. Quartan and malignant tertian together in 10 cases, 6 showing erythrophagocytosis. Quartan alone in 8 cases, 2 showing macrophages. Benign tertian 6 cases, erythrophages in one. Benign tertian and quartan together in 2 cases, with erythrophages in one. Malignant and benign tertian, one case; all three species, one case; in both ingested red cells were found. Malignant tertian alone in 3 cases, but no macrophages were seen in these. Erythrophages were found in one case in which no parasites were detected.

Phagocytosis occurred in 13 cases; vacuolated mononuclears were present in 28, in 3 of which no parasites were seen. It is in this age-group that the only 2 cases of associated auto-agglutination and auto-phagocytosis were observed.

Children æt. 3 years.—In 40 out of 42 cases malarial parasites were seen, and auto-phagocytosis occurred in 16. 7 out of the 42 were brought to hospital suffering from "fever." Benign tertian parasites alone in

11 cases: macrophages in 4. Quartan 16, with red-cell ingestion in 7. Malignant tertian 8: 4 showing erythrophages. Benign and malignant tertian, and quartan and malignant tertian, were each found in 2 cases; and all three together in one case: only this last showed macrophages.

Vacuolated cells were noted in 39 of the cases. An erythrocyte actually within the substance of the nucleus of the phagocyte was observed in one case in this group; in one case also was seen a phagocytosed infected red cell.

Children æt. 4–6 years.—Out of 135 blood-examinations, malarial parasites were detected in 127. Only 2 of the cases were obviously suffering from malarial fever. Erythrophages were seen in 34 cases and vacuolated large mononuclears in 118. 82 of the children were attending school at the time of examination, and these furnished 27 examples of auto-erythrophagocytosis. 52 quartan; 15 showing ingested red cells. 31 benign tertian, only one showing phagocytosis. Malignant tertian 12, erythrophages in 2. Quartan and malignant tertian together 14, macrophages in 6. Quartan and benign tertian parasites in 11 cases, phagocytosed red corpuscles in 8. Benign and malignant tertian in 6, macrophages present in one. All 3 species of parasite in one case only, and in this erythrophages were seen.

Children æt. 6–10 years.—In this age-group 103 examinations were made. In 90 malarial parasites were seen. In 27 of these macrophages, and in 83 vacuolated mononuclear cells, were seen. 78 of the cases were in children attending school, and only 4 were cases of obvious malarial fever. 46 quartan, phagocytosis in 12. Benign tertian 18, erythrophages in one. Malignant tertian 12, macrophages in 4. Benign tertian and quartan together 7, erythrophages in 5. Malignant tertian and quartan 5, auto-phagocytosis in 3. Malignant tertian and benign tertian 2, erythrophages being found in both cases.

Adolescents æt. 10–20 years.—In 15 out of 27 examined, malarial parasites were found; 6 quartan, macrophages in 2. 7 malignant tertian, macrophages in one. In 2 cases quartan and malignant tertian parasites existed together—no erythrophages were seen. Erythrophagocytosis occurred, therefore, in only 3 of the cases. Vacuolated cells were found in 17.

3 cases came to hospital complaining of body-pains and fever: no parasites were found in their blood. In this group occurred the second instance of ingestion of an infected red cell.

Persons æt. over 20 years.—33 routine blood-examinations were made at the hospital in patients æt. from 20 to 45 years. Most of them came complaining of "fever" or of "rheumatism." Malarial parasites were found in 9; quartan in 6 and malignant tertian in 3. Macrophages were seen in 3 of the cases, one of these showing malignant rings. Parasites were not seen in the blood of the other 2 cases, both of whom had cervical adenitis. Vacuolated cells occurred in 18.

Auto-erythrophagocytosis in malaria apparently tends to be more common in the earlier years of life than later.

Of the unmixed infections, quartan malaria shows the highest percentage of cases with macrophages, while benign tertian shows the lowest. Mixed infections, especially when all three types coexist, show a very high proportion of cases with ingested red cells. Observations in 22 cases showed marked effect of quinine on the phagocytic process; a dose of the drug hardly sufficient to destroy all the parasites usually resulted in the cessation of phagocytosis. A suitable dose usually caused the disappearance of the macrophages within three or four days. 13 cases of malaria were selected, in which quinine was withheld. The children were carefully watched and daily blood-examinations made. The results proved that the parasites may disappear from the blood stream without the aid of any drug. Erythrophages, present in several of these cases, disappeared coincidentally with the dying out of the parasites. Where signs of phagocytosis existed, evidences of anæmia were always present—*e. g.*, pallor, polychromasia, stippling of red cells and alterations in their size and shape. Normoblasts were also frequently observed.

Blood of Europeans.—The blood of 27 residents in West Africa was examined. 13 were apparently in health; in these, parasites were found in one only—three crescents. Other 9 were suffering from fever, and malignant rings more or less abundant were found in 7. No erythrophages were found in the blood of any of these 7, but vacuolated cells were seen in 6. Two cases were of quartan malaria. In one erythrophages and vacuolated mononuclears occurred. There were 2 cases of syphilis in the acute stage, with fever and abundant rash. Neither showed any macrophages or blood-parasites, but vacuolated cells were present in one. The list also includes one case of acute anthrax infection of the throat, one of acute axillary lymphadenitis, and one of an acute abdominal condition. Macrophages and vacuolated cells were not found in blood-films from these cases.

Smallpox Cases.—Films from 20 cases in different stages of the disease were examined. In one case only, a child, were macrophages seen; in this there were numerous benign tertian rings, and the malaria was probably the cause of the phagocytosis. No blood-parasites were seen in any of the other cases. Vacuolated cells were observed six times, but in all of these there were pigmented leucocytes, evidence of recent malarial infection.

Cases of Filariasis.—23 cases showing filarial embryos were examined, and 3 others with well-marked elephantiasis of the leg, in whom no filarial embryos were detected. There were 4 cases of *F. loa*, 4 of *F. bancrofti*, 5 of *F. perstans*, 4 of *F. bancrofti* and *F. perstans* associated, and 4 of *F. loa* and *F. perstans* together. In view of the frequent co-existence of trypanosomes and *F. perstans* in the human subject, it is noted that no macrophages were observed in the blood of any of the cases included in this group. Vacuolated cells were seen in 5 instances only. One of these cases showed an enormous number of *F. loa* embryos, and the vacuolated mononuclear cells were numerous.

Yaws, 10 cases; malarial parasites were found in the blood of 9. No erythrophages occurred in any, but vacuolated cells occurred in 9.

Beri-beri.—Of 6 cases, blood-parasites—embryos of *F. perstans*—were found in 2. Erythrophages were not seen, but vacuolated and pigmented mononuclear cells occurred in all.

Yellow Fever.—3 cases (Accra) were examined post-mortem. No parasites, macrophages, or vacuolated cells were seen.

Ankylostomiasis.—21 cases were examined whose fæces contained abundant ankylostome ova. Blood-parasites were found in only one instance, the infection being with scanty quartan rings; in this case erythrophages were absent, but were found in 8 of the 21 cases. Vacuolated mononuclears occurred in 17 cases and pigmented leucocytes in all.

In view of the prevalence of ankylostomiasis in West Africa, it was attempted to eliminate possibility of its being the main cause of the auto-erythrophagocytosis observed in malaria and trypanosomiasis. For this purpose 9 prisoners, in whose fæces numerous ankylostome ova occurred, were chosen. Macrophages were found in the blood of six. One of these showed a high eosinophilia. After treatment with thymol, the blood was examined again nine days later—macrophages had disappeared and only a few vacuolated cells were seen. At this time very few ankylostome ova were seen in the fæces. He received no quinine.

The remaining 8 prisoners showed only a slight eosinophilia. Macrophages were present in the blood of 5, and vacuolated cells occurred in all. All were given thymol treatment. Four days later the blood was re-examined. Macrophages persisted in 4 and were absent in 1.

The 4 prisoners still showing signs of phagocytosis were then treated with quinine for three days. Four days later, macrophages were still present in 3, and had disappeared in 1. Thymol alone caused disappearance of macrophages in 2 where quinine had not been given. The 3 cases which were unaffected by either quinine or thymol showed ankylostome ova in the fæces from first to last. It therefore seems possible that auto-erythrophagocytosis occurs in ankylostomiasis.

Conclusions.—Auto-erythrophagocytosis occurs in trypanosomiasis and in malaria. The phagocytic process was noted in some cases of trypanosomiasis, not in others. In the malarial cases, phagocytosis was observed in some mild infections, while it was absent in several cases where the parasites were very numerous.

In malaria, onset of phagocytosis is influenced by age of patient and duration of illness. It is more marked in quartan and malignant tertian infections that in benign tertian, and is more frequent in mixed than in single infections.

No adequate explanation of the origin of the process has been supplied. The presence of haemopsonin is doubtful. Experiments made with the first case of trypanosomiasis indicate that there is no opsonin; analogy suggests, however, that there is an opsonin. Auto-agglutination, well recognised and frequent in trypanosomiasis, has also been seen in malaria. A suggestion is here made that the erythrophages observed in the present series of cases are not normal constituents of the blood in the peripheral circulation. Effete red cells are said to be destroyed in liver and spleen. Smears made from these organs of slaughtered cows suffering from trypanosomiasis, and of a guinea-pig, showed erythrophages. Both liver and spleen are enlarged in trypanosomiasis and in malaria.

It may be that the erythrophages originate from these or other organs and under stress of work venture forth into the general circulation; the stimulus for this process may be the greatly increased number of damaged red cells resulting from the protozoal infection.

The disappearance of the phagocytes from the peripheral blood may be a natural sequence of adequate treatment directed against the parasite.

The theory which accords best with the facts is that the phenomenon is connected with the acquisition of immunity. The occurrence of erythrophages in some cases of mild infection and the absence in some cases of severe infection, their more frequent appearance in the early than in the late years of life, and the results in the cases which were not treated with quinine, are the basis of the theory.

It is fairly certain that children in West Africa are not born with any appreciable degree of immunity. Adult natives, on the other hand, are apparently immune to the effects of the parasite. The question arises: "Is it wise to dose all native children who suffer from malaria with quinine"? If quinine be given, the parasites are quickly destroyed and there is no opportunity of gaining immunity. It may be possible, after further research, to say that the presence of macrophages indicates that the child is able to deal with the parasites and is acquiring immunity, and that quinine is indicated in severe infections when there is no evidence of auto-erythrophagocytosis.

With Dr. Connal's interesting and valuable paper are given excellent photomicrographs illustrating erythrophages and the process of phagocytosis.

H. M. H.

ABSTRACT FROM 'JOURNAL OF EXPERIMENTAL MEDICINE,' Vol. xvi. No. 4.

THE CULTIVATION OF MALARIAL PLASMODIA (*P. VIVAX* AND *P. FALCIPARUM*) IN VITRO.

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IN November 1911, Bass reported the successful cultivation of *Plasmodium vivax*, *P. malariæ*, and *P. falciparum* in vitro. It was stated then that the incubation-temperature must be 40° C. or higher, strict anærobiosis seemed to be necessary, and the most favourable medium defibrinated or citrated human blood. Further experimentation along this line was begun in April 1912, when the Tulane School of Tropical Medicine equipped and sent to the Canal Zone a research party for the study of malaria.

Laveran reported malarial parasites to be visible in hanging drop preparations for as long as ten days, but was not able to satisfy himself that the parasites multiplied or even kept alive. Conrado's claim to have cultivated malarial plasmodia from infected water has not been confirmed. The authors' experiments, on the other hand, disprove such claim, for they find the parasite cannot live for any length of time in a non-isotonic fluid, such a fluid as water being absolutely destructive to both the red-blood cells and the plasmodia. Miller also reported cultivation of the parasites. His experiments have been repeated, but his claim is unconfirmed.

Sakharov, Rosenbach, Blumer, Hamburger, and Mitchell kept malarial plasmodia alive for several days in leeches that had been allowed to suck the blood of malarial patients. In Hamburger's experiments the æstivo-autumnal parasites developed motile pigment.

In Blumer's case the parasites showed no amœboid movement. In both instances the parasites became peculiarly refractive and glistening in appearance. The authors consider these forms to be dead. No segmentation and rosette formation were noted even in eight days. Their cultures, however successful, must be considered as having been accomplished *in vivo* instead of *in vitro*.

The authors state that their present technique is imperfect, and includes steps which are more or less empirical: they will serve, however, as a basis for further development.

Technique.—The apparatus and material necessary for the cultivation of one generation only of parasites are:—(1) 20 c.c. all-glass syringe with coarse needle. One must prevent leaking and bubbling of air into the syringe, and strong capillary force and strong suction, which may damage host, cell, and parasite. (2) Defibrinating tube. Tubes one inch in diameter and of whatever length will be accommodated by the centrifuge. The tube is plugged with a cotton plug having a glass rod running through it and extending to the bottom. In place of the rod a glass tube may be used. To this is attached the needle by short rubber tubing. Blood is collected directly into the defibrinating-tube by sticking the needle into an infected vein. (3) Culture-tubes $\frac{1}{2} \times 5$ inches. (4) 1 c.c. pipette, graduated in hundredths. (5) Dextrose (Merck's) 50 per cent. solution in water. (6) Capillary pipettes and rubber bulb: 3–4-sixteenths of an inch glass tubing is the proper size from which to make these pipettes. The capillary tube should be rather large—fine capillaries destroy the plasmodia. The large ends of these capillaries should be plugged with cotton before sterilising. The following additional apparatus will be required if more than one generation of parasites is to be grown, or if it is desired to avoid the dead parasites which are present in the deeper layers of cultures containing a thick column of cells:—(7) Centrifuge, 800 to 2000 revolutions per minute. (8) Culture-tubes $\frac{1}{2} \times 5$ inches, with flat bottom, or with disc of pure white filter-paper supported one-half inch from the bottom of tube by a piece of glass tubing. (9) Plain pipettes, capacity 5 to 20 c.c., with rubber tube 2 feet long, mouth-piece, and pinch-cock. These pipettes are plugged before sterilising and are used only once on account of the difficulty of cleaning them.

For Cultivating one Generation only.—Blood is collected from patient's vein and is expelled directly into defibrinating-tube. The latter should be tilted to one side and care taken to avoid unnecessary exposure of blood to the air. One-tenth of a c.c. of the 50 per cent. solution of dextrose for each 10 c.c. of blood to be drawn must be placed in the defibrinating-tube before the blood is drawn. By gently stirring or whipping with the rod or tube extending through the plug, defibrination is effected. Bubbles caused by the whipping in of air must be avoided. Plug and rod may now be replaced by one from another tube of the same size. The defibrinated dextrose blood may be transferred to other tubes or incubated in the original tube. In any case, column of blood must be one to two inches deep. This gives a column of serum one-half to one inch deep above the cells and parasites when the latter have settled. Supernatant serum more than one inch deep has no advantage. When this is less than one-half inch deep the parasites often die before segmentation. The parasites live and develop at the top of the column of precipitated cells in a layer varying from $\frac{1}{50}$ to $\frac{1}{20}$ inch thick. All parasites beneath this layer die in from 2 to 20 hours—the time is dependent on factors not yet determined. Parasites in the thin layer at the top may be examined at any time by drawing a small quantity of cells from this layer by means of a capillary pipette. The pipette is passed through a flame to sterilise it, but must be allowed to cool thoroughly before using, a temperature of 45° C. to 50 C. killing the parasites. Such a temperature also alters the red-blood cells in some way, so that they are rendered permeable to the surrounding serum and this destroys the parasite. Tilting to one side of a culture-tube results in burying and killing the parasites in the thin top layer.

For Cultivating more than one Generation.—The leucocytes must be removed when the culture is made in order to avoid destruction of the parasites by them at the time of segmentation. Infected blood from the patient is centrifugalised sufficiently to force the leucocytes to the surface of the cells, avoiding unnecessary centrifugalisation. The supernatant serum is drawn off and put into culture-tubes. The column of serum in each should be one-half to one inch deep. Cells and plasmodia are carefully drawn from about the middle of the centrifugalised cells and planted at the bottom of the serum in the culture-tubes. Flat-bottom tubes are an advantage. One to two-tenths of a c.c. of cells in a half-inch tube make the thickest layer in which it is possible to get a homogeneous growth of parasite. The authors secured growth of all the parasites in about twice this quantity of cells in a half-inch tube by employing tubes with a paper shelf suspended in them. They are filled with serum at least ½ inch above the level of the support for the paper disc. The disc is then carefully tucked in place, after which the cells are placed on it. Even more satisfactory results with tubes filled ½ inch or more deep with freshly prepared human plasma. These are conveniently prepared by placing one-half to one inch of blood, immediately after it is drawn, in culture-tubes, and centrifugalising to throw the cells to the bottom before coagulation takes place. By continuing centrifugalisation until after coagulation occurs, a flatter surface is secured for the plasma. In such plasma prepared tubes, one-half inch or more of serum is placed, and then cells and parasites from the centrifugalised and defibrinated dextrose blood are carefully distributed over the surface of the plasma. Parasites in such leucocyte-free cultures develop, segment, and most of the merozoites enter new red-blood cells. These young parasites develop in the same manner as the first generation and sometimes reach segmentation. In one instance three successive generations developed in such a culture. More often the parasites begin to die out after the first, and especially after the second, segmentation. The cause of this has not been determined. To perpetuate the culture it is necessary to transfer a portion of the cells and parasites to a recently prepared tube containing fresh cells and serum. It is convenient to place the fresh serum in the culture-tube and to take up in a large capillary pipette a portion of the cells and parasites of the culture, and then about five times the amount of fresh cells. These are mixed in the pipette with air excluded and then carefully spread on the surface of the plasma, paper shelf, or bottom of the tube, according to the particular kind of culture-tube used. The transplantation should be done within 4 to 5 hours of the time of maximum segmentation, and therefore approximately every 48 hours for tertian and æstivo-autumnal parasites.

The authors discuss the many interesting observations made on the parasites in culture, and conclude this interesting paper with the following

Summary.—The asexual cycle of *P. vivax* and *P. falciparum* has been cultivated *in vitro* in human blood.

The parasites grow within red-blood cells, and there is no evidence that they can be grown outside of these cells. The parasites are destroyed in a very few minutes *in vitro* by normal human serum and by certain modifications of human serum. Leucocytes phagocytise and destroy malarial plasmodia growing *in vitro* only when the parasites escape from their red-blood cell-capsule or when the latter is perforated or becomes permeable.

Successive generations of *P. vivax* and *P. falciparum* have been cultivated *in vitro* by removing the leucocytes from the culture and by transplanting to fresh red-blood cells and serum at proper intervals.

The asexual cycle of *P. vivax* and *P. falciparum* cultivated *in vitro* does not differ from the same cycle growing *in vivo*. The sexual cycle has not been cultivated, though some evidence was obtained of the possibility of its accomplishment.

Plasmodium vivax and *Plasmodium falciparum* when grown in an identical culture medium and under exactly the same conditions remain distinct. They are separate and distinct species. In 29 cultures of æstivo-autumnal parasites many forms and sizes have been observed, so that evidence is supplied of the occurrence of different varieties of æstivo-autumnal malarial parasites. The so-called tertian æstivo-autumnal variety may be seen at the proper stage in all cultures grown from merozoites. The form and appearance of the same culture of plasmodia may vary greatly under different conditions, which are not necessarily destructive to the parasites. Their generation-period may vary from 30 hours (æstivo-autumnal) to 4 days (tertian) as a result of variation in the temperature at which they were cultivated. Sexual parasites grow in the cultures and are more resistant to unfavourable conditions than schizonts, often living several days after the latter die out.

Forms suggesting parthenogenesis have been observed.

H. M. H.

V.—PUBLIC HEALTH.

By Prof. R. T. HEWLETT, M.D., F.R.C.P., &c.

The Bacteriology of Leprosy.

By H. BAYON,
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Research Bacteriologist to the Government of the Union of South Africa.

The tinctorial and morphological entity called Hansen's "bacillus" bases its claim to be the etiological factor of leprosy on its constant occurrence, at one period or another of the disease, in all cases of leprosy where the clinical diagnosis presents no doubts or difficulties. It must be admitted from the very onset that a certain minimal proportion of cases, which from their clinical course and appearance, if not identical, are at least very similar to leprosy, do not show Hansen's "bacillus" in the lesions in the quantity and position we generally meet it in lepromas. These aberrant cases I will consider later on and try to find a satisfactory explanation for their abnormal features.

Though the whole evidence I am now bringing forward step by step fits in accurately with the observations of many other independent workers, conclusive proof will be the result of facts and experiments whose discussion must follow the settling of the initial problems.

It is quite to be understood that Hansen's "bacillus" was to be a mere tinctorial and morphological entity so long as we were unable to get it in pure culture, or more exactly to recognise its parent form as such when we did get it; and that accordingly its properties in relation to the tissues it invades should be the subject of a certain amount of controversy.

The term "bacillus" when applied to the organism of leprosy is evidently a misnomer, which has been sanctioned by constant use.

In the tissues (and I may add in cultures) Hansen's "bacillus" possesses a most varied morphology. In the majority of cases it is a short, plump or slender rod, which may be slightly thicker at one end and taper off at the other or end bluntly. Definite uniform rods are also seen, and between these and the pure coccoid shape all gradations of morphology can be detected in the juice expressed from lepromas, in which I have also seen definite, big, clubbed shapes. These variations in morphology, which place Hansen's "bacillus" together with the tubercle organism in the group of "mycobacteria," have been again lately insisted upon by Galli Valerio.

The similarity of leprosy and tuberculosis is an oft recurring and disconcerting observation. I will mention later on the well-known tinctorial and morphological resemblance amounting to practical identity of the etiological factors of both diseases, as also their tendency of producing under circumstances similar lesions in internal organs. We know that lupus and leprosy are clinically sometimes very similar, except for the fact that multiplicity of nodules and presence of nerve lesions speak in favour of leprosy. Extreme friability of tissue is generally only to be met with in lupus. Lepers react to tuberculine in a similar fashion as tuberculous patients, except that the febrile reaction can be delayed till ten, twelve, or even sixteen hours after the injection.

The chronic course of both diseases, their intractability, their low infective properties, under certain circumstances, are again points of resemblance.

Should an acid-fast rod met with in leprous regions not be present in the overwhelming quantities usually associated with leprosy, and should it, moreover, give rise to these tissue reactions usually met with in tuberculous disease, the tendency of those investigating the case would be to immediately consider the acid-fast organism not to be Hansen's "bacillus."

Now it is certainly not without importance whether a patient has leprosy or tuberculosis, so that no excuse is needed for the attention that has been paid to the distinction of the two "bacilli" by means of staining reactions.

I have carefully read the literature on the subject, and having tried the various methods advocated by those who have investigated the matter (including von Betegh and Yamagiva), I have come to the conclusion that no method of staining exists so far that enables us to distinguish with certainty an individual tubercle "bacillus" from the organism of leprosy.

All we can say is that, on the whole, Hansen's "bacillus" is more variable in its resistance to bleaching with mineral acids, once it has taken the warm carbol-fuchsine stain, than tubercle, and that, moreover, it is also capable, on the whole, of being more easily stained with cold carbol-fuchsine than Koch's "bacillus." That is as far as we can go. Individually you cannot, of course, distinguish the two morphologically, and in lepromas and cultures I have found single specimens of Hansen's "bacillus" whose warm carbol-fuchsine stain was quite as resistant to thirty per cent. nitric acid as the control human tubercle culture specimens. There can be no doubt that "acid-fastness," generally speaking, is a varying factor, not only in different cultures of various bacteria, but also in the different individuals of the same strain.

What are the typical features of the leprous lesions, whether met with in the skin or the organs of the individuals affected?

MacLeod gave a short summary of the subject at the last Leprosy Conference at Bergen, and if I may be allowed to add my conclusions to those of this Author we get a description of the following distinctive features.

In the Skin.—Presence of acid-fast "bacilli" in big groups or closely matted bunches. The tissue reaction is of the "granulomatous" type without the strong predominance of plasma cells met with in syphilis. Giant cells of the true Langhan's type, *i. e.* with a necrotic centre, are not present or possibly exceedingly scarce. No necrotic tissue is visible in non-ulcerated nodules, and the diseased tissue merges into the healthy surroundings without a sharp or clear line of demarcation. Lepromas appear to be richer in small capillaries than the corresponding tubercular lesions, where a tendency to sclerosis is noticeable in the small blood-vessels.

The matted bunches of bacilli I consider to have arisen partially through the choking up of lymphatics with swollen leucocytes, which have taken up the acid-fast organisms in such quantities that they have accordingly bulged out and vacuolized so as to form a plug in the lymph spaces. The endothelium of the lymphatics also takes a part in the process by englobing the "bacilli" in great quantity, but this process may possibly be secondary to the temporary choking of the lumen of these lymphatics.

Long before the protoplasma of the leucocytes and endothelial cells has vacuolated and got completely disintegrated, the nucleus has degenerated and slowly got eliminated, the remnants of the cell without its nucleus giving rise to the "globi" which have been so often described. In the course of time the last remnants of the protoplasma of the cells disappear, and the "bacilli" seem to be loose in the tissues and form under circumstances peculiar ramifications in what one may call connective tissue septa.

I have not been able to decide with certainty whether the curious hyaline coating which is seen on many "bacilli" in leprous lesions is secreted by the bacteria themselves, or is a product of the cellular protoplasma of the human body. I rather incline, however, to the latter view, not having ever noticed it in artificial cultures.

The presence of big vacuolated cells containing acid-fast organisms is sufficiently typical in itself, but as a whole the bacteriological and histological reasons for diagnosing a skin affection as nodular leprosy can be summed up as follows:—Presence of acid-fast rods in abundant quantity and in matted groups, absence of necrosis, caseation, or giant cells of the true Langhan's type in a granulomatous matrix.

These observations have been made not only on sections of leprous nodules, but also on the juice squeezed from such and from smears taken from excised bits. A good method of studying cellular alterations consists in dabbing a freshly excised nodule from which the oozing juice has been removed by sterile saline on to a series of slides. The adherent minute fragments of tissue allow examination with very high powers.

In the Inner Organs and Glands.—Every viscus of the human body is liable to be invaded by Hansen's "bacillus," but the ones most frequently noticed are the testes, liver, and spleen. An intestinal form of leprosy has been described to me by my co-worker Dr. Martinez, of Bogota, who considers it very similar in its naked-eye appearances to intestinal tuberculosis.

Generally speaking, however, we may say that, taking into consideration the cellular differences of each habitat, the lesions resulting in the inner organs are analogous to those of the skin, except that in these cases giant cells very similar to those of the Langhan's type may be present in small numbers, and a certain degree of necrosis or even caseation may make its appearance. The only remaining feature to distinguish these lesions from tubercle is the quantity of the acid-fast organisms present, but also this point may not allow a clear decision, so that the experiment with guinea-pigs may have to be resorted to, so as to enable a positive diagnosis to be made.

This fact has a certain bearing on the apparent liability of lopers to die of lung tuberculosis; the mortality due to this cause being put down by some as high as sixty per cent. of cases coming to autopsy.

Turning now to the bacteriology proper of leprosy, we find that Hansen's "bacillus" occupies an exceptional position. Though present in enormous quantities in the tissues, no culture medium has succeeded in getting the organism to multiply artificially to any extent in its original morphology and with the staining properties it has in tissues. The various cultures which have been published from time to time I will deal with separately later on. Not only did the bacterium of leprosy appear not to be artificially cultivable but it seemed to have a special biology of its own, for it was not transmissible from man to animals, and no disease was known in animals which presented analogous histological and bacteriological features to leprosy in man. I believe I am now in a position to modify all these statements, having cultivated Hansen's "bacillus" in its parent form and in its acid-fast phase, and having transmitted leprosy to rats in the second generation by injecting nodules taken from a leper. Regarding, however, the absolutely isolated position of leprosy as a disease peculiar to man only, Stephansky, George Dean, and Lydia Rabinowitch clearly demonstrated that a similar disease existed in sewer-rats in Odessa, at Elstree near London, and in Berlin. This disease has now been found in wild rats practically all over the world. A somewhat similar disease is Johne's pseudo-tuberculosis of cattle, which presents several of the peculiarities of leprosy when attacking the intestinal tract ; and quite recently Borel has been studying another similar affection in horses. I have no doubt that leprous diseases are present in many of the higher mammals, but they do not present the marked dermal features of the disease in man, and therefore are often confused with tuberculous affections.

The recalcitrant attitude of Hansen's "bacillus" towards artificial cultivation is absolutely incomprehensible, unless we admit that it is not a real "bacillus," but simply the acid-fast stage of a filamentary, branching organism which is absolutely non-acid-resisting, but which at certain phases of its existence can break up into acid-fast fragments. As a matter of fact, to anybody who has worked with these branching non-acid-resisting or partially acid-proof organisms, Hansen's "bacillus" is immediately recognisable by its morphology in leproma juice as an acid-fast diphtheroid, and accordingly as the fragments of an "actinomycotic" germ.

Once it is admitted that Hansen's "bacillus" is but the end phase in the tissues of a filamentary and branching organism, a quantity of bacteriological and also clinical observations find an easy and satisfactory explanation.

The cultures of Bordoni Uffreduzzi, Babes, Kedrowsky, Levy, Czaplewsky, Raki and Buckmaster, Spronk, Shiga, Hewlett, and others are shown to probably or possibly have been leprosy, though at the time and up to the present day they may have earned but very scanty recognition.

As it is well known, all these authors have cultivated from the nodules and inner organs of lepers (and in some cases repeatedly) non-acid-resisting organisms of a branching, filamentary type, or more often slightly resisting bacteria of diphtheroid morphological characteristics, which they believed were cultures of the organism of leprosy. It must be added, however, that only in one case—viz,, Kedrowsky—was the author able to bring forth experimental proof of his views. It was only Kedrowsky who, having the time, patience, and perseverance to continue his experiments for ten years, succeeded in producing in rabbits and mice, by injecting his leprosy cultures, lesions comparable to those occurring in human beings and in regaining his organisms in an acid-fast constant stage. Not only this, but he succeeded in isolating in pure culture the organisms he had injected into his experimental animals as non-acid-fast bacteria, as absolutely acid-fast rods identical in all particulars with Hansen's "bacillus" in lesions.

This is confirmatory evidence in favour of the view that the organism of leprosy belongs in reality to the class of actinomycotic organisms. Such a revolution in our views of the stability of bacterial species cannot be straightaway accepted without further confirmation or the knowledge of analogies from independent sources. Leaving momentarily aside the work I have carried out confirming the presence of a filamentary, branching, non-acid-fast parent form of the organism in leprous lesions, attention can be drawn to the big group of ubiquitous germs, partly pathogenic and partly innocuous for animals, which have been discovered in the most varied localities by different and numerous workers. These branching, filamentary organisms have been shown to be able to break up into acid-fast fragments ; in fact, in some rare cases these fragments have been regained in pure culture as acid-fast rods, and have been known to reproduce in animals lesions very similar to those of tuberculosis. I mention at random the *Actinomyces capræ* of Silberschmidt, Birt and Leishman's streptothrix from man, Grasberger's "bacillus" from butter, the filamentary acid-fast organism observed by Flexner in a negro, Nocard's "farcin du bœuf," Sanfelice's experiments with *Actinomyces alba* in dogs, Rost's so-called *Streptothrix lepræ*, and Deycke's organism. Birt and Leishman's case may serve as a paradigm of the whole group.

A soldier returning from South Africa presented the clinical features of tuberculosis of the lungs and showed acid-fast bacilli in his sputum. He developed pleuritis, and out of the fluid a non-acid-fast, branching, filamentary organism was repeatedly cultivated. On autopsy the lungs showed lesions somewhat similar to tuberculosis, but fragments inoculated into guinea-pigs did not give rise to tubercular disease.

Birt and Leishman's organism is capable of breaking up into acid-fast fragments in artificial cultures, in the same way as the other bacteria I have already mentioned also do. I need not further labour this point, an able discussion of which is to be found in Foulerton's Milroy Lectures.

The clinical similarity of leprosy with skin-diseases caused by acknowledged actinomycotic organisms has been often noticed, quite lately again by Galli Valerio. These are, however, quite minor points; it is sufficient to show that the fact of Hansen's "bacillus" being the acid-fast phase of a non-acid-proof, filamentary, branching organism does not stand alone, but that there are, as is to be expected, quite a number of analogous bacteria with similar well-marked properties.

The observations of the various workers who cultivated filamentary organisms and diphtheroids from leprosy find therein a satisfactory explanation, and, moreover, it shows once more that the main difficulty in connection with the bacteriology of leprosy consists in the identification of the organism isolated from the patients and not in the culture of the parent forms, which I believe can succeed on many different media.

Having already described my own results of the attempts to cultivate Hansen's "bacillus" in various previous papers, I will simply briefly recapitulate them once more.

On two occasions I isolated from the nodules of a leper at the London School of Tropical Medicine an acid-resisting diphtheroid. By acid-resisting I mean that after staining for half an hour with warm carbol-fuchsine it could not be bleached by 1 per cent. sulphuric acid in five seconds. This diphtheroid was mixed on one occasion with an absolutely acid-fast rod, which resisted Ziehl-Neelsen's method of staining. I only followed, however, the behaviour of the pure diphtheroid culture. This diphtheroid was shown by Dr. Priestley, of the Lister Institute, by means of sugar tests, to belong to a separate group by itself and not to be confused with any of the common diphtheroids found on the surface of the human body.

On injection into the peritoneum of a mouse it acquired acid-fast properties in the abdominal glands without markedly changing its morphology; when regained in pure culture it resisted bleaching with 25 per cent. sulphuric acid. On another occasion I isolated in pure culture from the nodules of the same patient a filamentary, branching, absolutely non-acid-resisting organism, which grew quite rapidly within three days when subcultured on the culture medium used. It was a milky-white moist growth. This I injected into rats, and found after three months that it had turned acid-fast in the nodule that developed at the site of injection. Subcultures taken showed an acid-fast rod identical in morphology with Hansen's "bacillus." The resulting growth was practically identical in all details with the moist, wrinkled, milky-white, acid-fast culture isolated by Kedrowsky in Moscow, only that it took much longer to grow in subculture, in some cases as much as six months.

In some way my work so far is a complete confirmation of Kedrowsky's work and agrees completely with the opinion expressed by Beauchamp Williams regarding the nature of the parent form of Hansen's "bacillus."

The medium used for the isolation of these organisms was placental-juice-glycerine agar. I have also found horse-serum-nutrose agar to be very good for subcultures, but believe that many other culture media exist and can equally well be made use of, the main difficulty lying in the identification of the organisms isolated, and not in their culture.

The next point that requires elucidation is the question of the transmission of leprosy to animals directly from the human being. To my way of thinking this has been successfully achieved on a few occasions, as there is no doubt that the process takes a very long time in completion, and failures and partial results are numerous. I believe that in the light of our present experience we must admit that the experiments of Melcher and Ortmann, Wesener, Wnoukow, Stanziale, and Thiroux, with rabbits and monkeys, were crowned with success, and would have been admitted as positive results if the unlucky phrase had not been coined that these authors had sown leprosy and reaped tuberculosis. As it is, the lesions caused by tubercle, human and avian, in rabbits are very similar to those brought about by the injection of my culture and also Kedrowsky's culture of leprosy, both of which I know for sure, after experimenting with guinea-pigs and fowls, are not human or avian tubercle.

In the 'British Medical Journal' for February 1912 I have given a short survey of the literature on the subject of the transmission of leprosy to animals by the injection of "virus" from lepers. I announced then the first localised lesions I had got by injecting the ground-up nodules of lepers into the testes of laboratory white rats. The animals were killed after five months, and acid-fast rods were only found at the site of injection and in the corresponding inguinal glands. These I injected into other rats, which I killed five months after and found numerous deposits of acid-fast rods in the spleen and liver. The quantity was by far superior to what had been injected, and the position in abundance, morphology, and staining reaction of the "bacilli," together with the histological appearances, satisfied me that leprosy had been successfully directly transmitted in the second generation. No giant cells of the Langhan's type were present; no caseation or necrosis was visible; round-celled infiltration was absent. The hepatic cells and the endothelium of the lymphatics in the liver and spleen had taken up the bacilli in big quantities without necrosing, though several were vacuolated and swollen to a marked extent. In the hepatic cells a brown, dark, coffee-coloured pigment was present in many of the cells invaded by the leprosy organisms; and it is worthy of note that this condition of cell vacuolization, in conjunction with the production of pigment and of course the presence of acid-fast rods, is typical of leprosy in the human liver.

In the spleen the "bacilli" had been mostly taken up and had multiplied in big mononucleated cells without causing the nucleus to disintegrate or even to stain faintly. No giant cells were present; in fact the usual passive quiescence of Hansen's "bacillus" was once more clearly demonstrated in these sections.

There cannot be much doubt that we are dealing with leprosy in these cases, especially as the microscopical appearances confirm so completely those produced by the injection into rats of the ground-up nodules of spontaneously leprous rats.

This brings our attention to bear on rat leprosy, a disease which is a complete analogy of leprosy in man, and from which George Dean cultivated a diphtheroid non-acid-fast organism which gave typical reactions with the serum of lepers.

This disease has lately been carefully studied by Marchoux, who has found that about five per cent. of the sewer-rats in Paris are infected by it. He also found that, though it is relatively easy to produce a localised deposit of the "bacilli," it is very difficult to cause microscopic lesions of the internal organs, and so far has not succeeded in getting experimentally the skin lesions which are the outstanding feature of the spontaneous affection in rats.

These results agree absolutely with the protocols of the experiments I carried out with the culture isolated from a case of rat leprosy.

From a nodule resulting in a rat injected with ground-up leprous lesions of a spontaneously affected rat, a white moist creamy growth was isolated which, under the microscope, showed itself to be a pleomorphic absolutely acid-fast rod indistinguishable from the culture isolated from a leper. This culture on injection into rats produced the identical visceral lesions which I have observed in rats injected with the virus from spontaneously infected animals; these lesions are produced again by an enormous quantity of acid-fast rods with little or no tissue reaction such as I have described in the corresponding human leprosy specimens.

So far this culture is only distinguishable from the human strain by the fact that it grows somewhat more rapidly, in fourteen days, on fish-juice agar, and is more pathogenic for rats than the human variety. If injected into fowls in big quantities (three culture slopes or more) it is capable of producing lesions not unlike those seen after injection of big quantities of avian tuberculosis. Such lesions I have so far not been able to produce in birds with the human strain of leprosy, because even big quantities were eliminated harmlessly by the fowls injected.

These are, however, but small differences, and this culture bears out again the statement that human and rat leprosy are closely allied, if not identical. We have similar conditions in human and bovine tuberculosis.

Surveying, however, the work carried out, we find that whether we inject into rats or mice the filamentary, branching, non-acid-fast organism cultivated from a leper, or the acid-resisting diphtheroid stage of the same organism, we get, if sufficient time is allowed, lesions identical with those resulting from the injection of the acid-fast rod stage of ultimate cultures. These sections are again identical in appearance and detail with the lesions resulting in rats after injection of ground-up nodules from a patient (virus). By injecting into a rat the culture of rat leprosy we get the same visceral lesions of the spontaneous disease, which are also identical with those resulting from the injection of virus (ground-up nodules) of naturally leprous rats. These lesions present a most striking similarity, amounting to practical identity, with the results of injection of various stages of human leprosy cultures and of human leprous virus. The whole evidence seems to me to correlate satisfactorily.

In my previous paper I mentioned that I could not explain the fact that on attempting cultures from the human leper we appeared to get with great ease the actinomycotic and diphtheroid stages of Hansen's "bacillus," but that once it had reached its acid-fast stage in experimental animals it was constantly regained as an acid-fast rod. This missing evidence has now been partially furnished by experiments carried out in the course of the last year. On two occasions non-acid-fast, filamentary, branching organisms were regained from animals (a mouse and a rabbit) which had been injected with the acid-fast stage of human leprosy culture. One of these isolations was carefully followed up, and now, after fourteen months' culture on Dorset's egg medium at 37° C., it has been found on examination to have turned into an acid-fast rod. The change has of course taken place gradually, and in the beginning the rods were nothing more than little acid-fast granules which appeared to gradually increase in size, and now, after fourteen months, a fair number of acid-fast "bacilli" are mixed up with the original culture. The identification of this organism has not been so far completed, but it has been most interesting to note the acquisition of acid-fast properties by a non-acid-fast organism on an artificial medium.

Regarding the cultures of Duval, Rost, and Clegg, I regret very much to say that in the repeated experiments I have carried out with them, by injecting them into monkeys, rabbits, rats, and mice, in no single instance have I been able to produce lesions resembling leprosy in any fashion whatever. Control animals injected with common saprophytic acid-fast organisms, such as Moeller's "dung bacillus" or Grasberger's acid-fast from milk, showed under such circumstances lesions identical with those produced by the intravenous injection of big quantities of Duval's and Rost's cultures.

Culturally they also behave as saprophytic acid-fast organisms because they grow at room temperature on common gelatine, and can be regained with the greatest ease on common agar from the organs of the animals injected.

This, however, is mere negative evidence. If Duval and Rost claim that their cultures are true leprosy they should publish definite and conclusive evidence that their organisms are capable of producing on injection into animals lesions that can be compared with those occurring in lepers or resulting in animals after injection of lepra virus. This is the case with rat leprosy, and has repeatedly been demonstrated to be feasible with the cultures I have isolated from a leper. So far, however, Rost, Duval, Clegg, and Williams have only communicated results that could be brought about by any acid-fast organism of the big saprophytic group.

So as to condense the subject matter of my paper I have not given extracts from my protocols. All my animal experiments have, however, been repeatedly carried out; it may be sufficient to say that to date they number over two hundred. The culture media tubes used are well above twelve hundred. I have, of course, a corresponding number of microscopical sections.

Before concluding I should like to mention the serological tests carried out with various cultures.

I can now report on tests carried out with the serum of five lepers. Their origin was as follows: one case from Mauritius, one indigenous case from England, one from the West Indies, and two from India. Three normal sera were used as controls. The test used was the complement absorption method, because on the whole agglutination has proved rather unsatisfactory with acid-fast organisms.

The antigen was prepared by alternately freezing and thawing with prolonged shaking of the culture emulsions used. The sera employed were diluted in falling doses and linked up with full, $\frac{1}{2}$, $\frac{1}{4}$, and $\frac{1}{8}$ quantities of titrated antigen according to a modification of the method of H. R. Dean which has already been fully described.

The general results were that the serum of some lepers contained a fair amount of antibodies, which enabled them to absorb complement in dilutions up to $\frac{1}{80}$–$\frac{1}{120}$ when linked up with an antigen made with one of Kedrowsky's or the Author's cultures. The same serum was practically negative towards Duval's and Rost's cultures, and gave modified, *i. e.* incomplete, reactions with human and avian tubercle antigen. Other sera of lepers absorbed complement to a much smaller extent ($\frac{1}{30}$–$\frac{1}{40}$), and others were practically negative ($\frac{1}{5}$–$\frac{1}{8}$). These, however, are absolutely negative towards Rost's and Duval's cultures, so that Kedrowsky's culture showed in every case if not a typical at least a slight reaction.

The normal sera were absolutely negative as is understood because the antigens were titrated against normal serum—that is, an initial dose of antigen was used that not only did not absorb mechanically but also did not react with the test serum. It will be seen that in no case did Rost's or Duval's cultures give any reaction worth mentioning; what they gave was simply group reaction probably due to their acid-fast coating.

Incidentally I may mention that Dr. Ledingham, of the Lister Institute, carried out some tropin-contents estimations with leper sera. He found that leucocytes treated with inactivated leper serum took up Kedrowsky's culture to a very marked extent, whilst controls with tubercle and Duval's culture were practically negative. The proportion in one case was like 2–7 in favour of Kedrowsky's culture.

Similar conclusions were arrived at by Dr. Hunt, of St. George's Hospital, by means of the opsonic index method. In this case Kedrowsky's culture was tested against human and bovine tubercle, and the serum of a leper and a tuberculous patient were made use of.

A bacterial extract has been made from these cultures on the same lines as Koch's alt-tuberculine. It was found to give rise to a typical feverish reaction in the two cases in which I was able to employ it. The nodules reddened and got inflamed after repeated injections; the lips got swollen. The symptoms passed off as soon as the injections were stopped.

Dr. MacLeod saw marked improvement in one case in which it was used, after prolonged treatment with Nastin had had no effect. He has reported on this patient in the 'Proceedings of the Royal Society of Medicine,' 1912, vol. v. pp. 142-144.

My own personal experience is of too short duration to be of any practical value. During a course of injections a definite but slight improvement resulted in the case treated, the disease progressing again, however, as soon as the injections were stopped.

The results are, however, encouraging, and I am going now to repeat them on an appropriate scale in South Africa, especially with a view of enabling an early diagnosis of the disease.

RAIN AS A DRINKING-WATER SUPPLY IN BRITISH GUIANA.

BY K. S. WISE, M.B., B.S., B.Sc. (Lond.), D.P.H.,

AND

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THE City of Georgetown, British Guiana, lies on flat alluvial land below the level of spring-tides, in the angle between the Demerara River and the sea-coast. For drinking-water it is dependent almost entirely on rain-water, the general average rainfall being about 100 inches per annum.

There is another water-supply, instituted mainly for sanitary purposes and for extinguishing outbreaks of fire. This water is rain which has fallen on an empoldered area of 70 to 80 square miles. This area is flat clay soil covered with heavy tropical growth of all kinds and littered with decaying vegetation, in places many feet thick. On this peaty material lies the conserved water which, in the course of months or even years, absorbs organic, inorganic, and colouring matters of all kinds. The water is brought down through an open canal to the middle of the city, whence it is pumped through an iron pipe system to taps and standpipes.

The water is of a light brown sherry colour, quite clear, and has no objectionable taste. It is, of course, a very soft water, having about three degrees of hardness, taking up lead readily and rusting iron pipes. It is slightly acid and has a high albuminoid ammonia figure, not so much by reason of fæcal pollution as because of organic solids held in colloidal solution.

This water-supply, while bacteriologically pure at its source in the conservancy, is open to frequent and intermittent pollution during its transit in the canals. Furthermore, the pipes conveying the water to all parts of the city are laid in soil which has received the overflow and soakage of numerous cesspits for many decades, so that a false joint, a negative pressure, and suction in the pipes may lead to large and dangerous contamination.

The result of bacteriological analyses conducted on water taken from the taps in Georgetown shows that this water after passing through the pipes is not fit for drinking.

The rain falling on the roofs is collected by gutterings which converge to large pipes leading the water into wooden vats. Each house has from one to four vats comprising a capacity of 1500 to 20.000 gallons.

The roof is generally of wooden shingles, occasionally of galvanised-iron sheeting, patent tiles, or slates. These wooden shingles are made from wallaba wood, and when first placed on the roof give up to the rain-showers a certain amount of colouring matter, tinging the water pale brown. It may be several months before this pigment is all extracted.

The great majority of storing-vessels are wooden vats of capacities ranging from 1500 to 8000 gallons each. These vats are placed in the open alongside or behind the houses, supported on timbers 6 to 8 inches off the ground. Occasionally the vats are placed under the houses, which are built on 10-foot pillars. A tap is fixed so as to draw off the water 6 to 9 inches from the bottom, and if two or more vats are supplied to a house they are usually connected by pipes so that water can pass from one to the other. In many cases there is a cistern near the roof into which the water is pumped from the vats, so providing a pipe-and-tap system over the premises as well as a bath shower.

Previously to 1909 most vats were supplied with a wooden cover, which rested on transverse beams across the top, allowing a space of 8 to 10 inches all round. Since that date anti-mosquito regulations have been enforced, and either the space is covered with mosquito-netting all round or the top is dropped down on to the rim, a square hole cut covered with netting through which the collecting pipe from the roof directs the water.

No measures are adopted (other than the above netting recently introduced to prevent mosquitoes breeding in the vats) to remove from the incoming water vegetable, animal, and mineral impurities off the roof. Of the many separating devices for rejecting the first washings off the roofs, few or none are used. The Roberts & Gibbs' rain separator has been tried but is very liable to rust, and thus is found fixed and useless at the time when most wanted. In some cases separators turned over by hand have been installed, but are soon forgotten and are left unused.

The typical wooden vat is in many instances replaced by iron tanks or iron cisterns of all shapes and sizes. They are usually built on a concrete base and have sloping roofs. In a few cases old stone cisterns are used. These are usually very old, having been constructed many decades ago of brick, of concrete, or both. These cisterns are wholly or half submerged in the ground, and receive by pipes the rain falling on a neighbouring roof.

Some of these cisterns have been shown to contain dangerously polluted water; a small fault in the concrete or brickwork has permitted the surrounding water to enter, bringing with it the overflow from cesspits and privies as well as the drainage of a sewage-saturated ground.

In the poorer parts of the town the rain-water is caught off the roof in a similar fashion and run into small iron tanks (400 gallons), barrels, tubs, and pails.

Efforts are used by some to sterilise the water by boiling, but with native cooks there is grave doubt whether the boiling is constantly and efficiently carried out. In a few instances Berkefeld filters have been used, also various filters made of compressed asbestos, wool, &c., all of them quite useless.

Rain-water has also been used on certain of the estates growing sugar-cane, where a population of 2000 to 5000 people has to be supplied.

In one case (Blairmont) the roof of the factory building has been utilised and a large concrete reservoir of 200,000 gallons built.

This reservoir is protected by galvanised sheeting all round, and the water is pumped from this reservoir to small subsidiary iron tanks outside, from which the water is drawn through taps.

At Providence, Demerara, a canal has been dug about ¾ mile long and containing many million gallons. This canal is filled by the rain falling directly into it and on the watershed formed by sloping its banks for 10 to 20 feet each side.

A subsidiary amount of drinking-water is supplied on this same estate by utilising the roofs of the ranges in which the labourers live as a watershed and collecting this rain-water in large iron boilers (30,000 gallons each) placed between the ranges.

At Springlands the roof of the factory building is used as a rain-collecting area, from which the water is run into a wide deep trench with a capacity of many million gallons, from which the water is dipped out as required.

Rain-water approaches nearer the absolute purity than any other kind of natural water, but quite apart from the uncertainty of the rainfall from year to year, the unusual length of the dry season at occasional periods, and the large size of the reservoirs thus required, there are other drawbacks to rain as a general source of domestic supply.

To quote a recent authority on Hygiene (Notter and Firth, 1908) :—"Rain-water as ordinarily collected and met with is an impure and dirty water, and its use as a supply for drinking purposes only justified in places where no better source is available. The chief merit of rain-water is its softness, owing to the absence of salts of lime and magnesia ; on this account it is good for washing and cooking purposes, although this very attribute of softness renders it less palatable than other kinds of water for drinking."

The system of collection adopted in this country allows many opportunities for the addition of foreign substances.

The rain in falling through the air washes out soluble substances, such as ammonia and nitrates (products of vegetable and animal decomposition), also chlorides, due no doubt to the proximity of the sea. Insoluble materials, such as soot and vegetable débris, floating in the air are also carried down.

An extremely valuable series of careful analyses carried out for over 20 years by Professor Harrison, the Director of Science and Agriculture, shows the quantities of ammonia, chlorides, and nitrates collected by rain falling in the vicinity of Georgetown.

In this series (see Table I.) the rain-water was gathered in the Botanic Gardens to the East of Georgetown through a clean glass funnel.

The customary collecting-area, the roof, gives opportunity for great and serious pollution.

Dust of all kinds, vegetable, animal, and mineral, is washed down into the reservoirs. The vegetable materials include pollen-grains, dry decayed leaves, twigs, straw, &c. ; the animal includes refuse from the roads and surface of the ground ; the mineral components are masses of angular grit of all kinds. The usual disposal of excreta is a cesspool, which in a large number of instances, more especially in the poorer yards, is nothing but an open pit. A little heavy rain fills this pit and floats over the surface of the yard (and of surrounding yards also) much of the human excrement contained within.

The subsequent drying of this surface and its resolution into dust shows the dust contamination of the roofs to be anything but a theoretical danger.

Birds, of course, find the roofs a suitable place to perform the major functions of life, and while in most instances this fouling of the roof is comparatively harmless, in the case of carrion-crows a direct and positive danger is always present.

These repulsive looking and repulsive smelling animals feed on most indescribable filth and carrion, and, as preliminary investigations show, harbour in their intestines actively pathogenic bacteria, such as *Bacillus dysentericus* and *Bacillus pyocyaneus* (see Table IX.). A considerable amount of the intestinal disorders of British Guiana is referable to one or other of these micro-organisms.

Furthermore, these vultures carry materials from the abbatoir from dead animals in the streets or yards &c. on to the roofs, and there dissect at leisure these repulsive and disgusting remnants. The guttering and piping is responsible for the possible addition of further materials to the rain-water (see Table II.). The gutters frequently sag, and form small pools of water with sometimes large quantities of decaying vegetable matters.

TABLE I.

Year.	Inches of Rain.	Milligrams per litre at 84° F.		
		Chlorine.	Nitrogen as Ammonia.	Nitrogen as Nitrates.
1890	125·63	8·93	·103	·178
1891 ...	109·66	5·04	·034	·131
1892	128·03	3·81	·044	·035
1893	135·24	3·15	·068	·066
1894	85·35	3·01	·045	·041
1895	82·56	4·19	·065	·237
1896	83·82	6·12	·044	·099
1897	98·84	6·06	·066	·059
1898	132·16	5·15	·058	·045
1899	52·72	7·69	·020	·093
1900	95·51	6·31	·019	·059
1901	75·97	7·48	·006	·028
1902	99·36	7·49	·041	·076
1903	108·58	5·68	·029	·073
1904	84·98	7·02	·045	·020
1905	75·66	6·58	·020	·074
1906	101·25	7·18	·007	·050
1907	117·65	6·05	·011	·028
1908	88·72	7·96	·015	·022
1909	105·76	6·35	·015	·070
Average 1890–1910	99·27	5·16	·018	·034

Mosquito larvæ and the eggs are often found in such places, and with a spate of water will be swept into the vat.

Rain-water is soft, and thus will take up lead and iron from the pipes passed through. As Professor Harrison's analyses show, traces of lead are sometimes to be found in rain-water in vats (see Table II.).

In the vat most of the impurities sink naturally to the bottom. Here is found a black slimy mud, frequently from 3 to 6 inches in depth, but in some stone tanks as much as 12 to 18 inches deep. Examination of this slime shows that it is almost wholly composed of mineral matter with numerous diatoms. The quantity of vegetable material which is washed in and sinks to the bottom undergoes a slow fermentation, supplying the lower layers of water in the vat with nitrogenous decomposition products. Consequently water drawn from the tap near the bottom shows a greater quantity of free and albuminoid ammonia than that drawn from the top.

Occasionally rats and mice have fallen in by accident, become drowned, and undergone decomposition in the vat-water. Rain-water conserved in iron tanks is liable to contain small quantities of iron. The water from brick and concrete cisterns built in the ground is looked on with grave suspicion, and is always liable to dangerous pollution. Latrines are frequently found situated close to them, and in several instances cesspits have been dug within 15 feet of such cisterns. These cesspits consist of a rusty old iron tank simply sunk in the ground, in some cases the bottom or a side being absent.

We are indebted to the Director of Science and Agriculture, Professor J. B. Harrison, for the following two tables (Tables I. & II.) representing a series of analyses performed by him on samples of rain-water during the last twenty years in this colony.

The average rainfall taken for the twenty years from 1890 to 1910 was 99 inches, the average chlorine content being 5·162 milligrams of chlorine per litre (·5162 parts per 100,000); the variation also being extremely great, the chlorine ranging from 1·7 to 37·0 as the two extremes.

The determinations of the chlorine and nitrogen in the rainfall of British Guiana have been carried out continuously for a period of twenty years, and are expressed as under in tabulated form.

These estimations were taken with great care, the rainfall being collected in a chemically clean glass funnel, so that they may be taken as a fair average of the rain-water as it falls from the sky. As will be seen, the nitrogen as ammonia is very high, and therefore this figure is of no value for future examinations as to pollution. The chlorine figure is equally valueless, as the city being close to the sea the rain naturally contains very varying amounts of chlorine.

We therefore have for some time now placed more dependence on the figure for phosphates as a chemical estimation of pollution in the absence of a bacteriological examination.

This is well shown in Table II. Sample No. 1 was collected in chemically clean utensils, but the succeeding samples, Nos. 2 to 9, were collected from vats in the ordinary way. The amount of phosphates present in most cases points to fæcal pollution. The danger of the plumbo solvency of this water is shown in the last column.

In 1893 a report was issued by the Government Analyst (Prof. J. B. Harrison) dealing with chemical analyses of the vat-waters used in various Public Institutions. These represent by far the best of the rain-water supplies as used in the city, as these buildings are kept tolerably clean and the vats cleaned out periodically at fairly regular intervals, usually from one to three years. Also the writer notes that the samples were taken from the clear water at the top without disturbing the sediment.

TABLE II.

The following are the results of chemical analyses of collected rain-waters from various sources in the city; in all cases the collecting area was the roof of a public building:—

No.	Total solids.	Chlorides.	Free ammonia.	Albuminoid ammonia.	Phosphates.	Lead.
1	2·1	·25	·0007	·0026	None.	None.
2	1·3	·272	·0005	·0007	Traces.	None.
3	3·6	·222	·0004	·0005	Traces.	Traces.
4	1·1	·421	·0040	·0005	None.	None.
5	·4	·148	·0002	·0040	Traces.	None.
6	2·5	·272	·0002	·0080	Traces.	None.
7	1·2	·297	·0048	·0048	Traces.	Traces.
8	2·9	·223	·0071	·0034	Traces.	Traces.
9	·2	·148	·0005	·0107	Traces.	Traces.

The figures represent grains per gallon at 80° F.

The microscopical examination of the sediment from clear water showed the presence of vegetable débris, spores, zooglea, infusoria, and cotton-fibres, with ciliated organisms occasionally.

Numbers 2 and 3 are the purest of those examined chemically, but the presence of phosphates in them and the results of the microscopical examinations show that even these are contaminated with filth, probably derived from the excrement of birds.

Table III. gives samples of rain-water taken by us from stored rain-water in use on various estates situated in the country.

As will be seen by these analyses the stored rain-water is liable to very serious pollution, especially when stored for considerable periods. The amount of filth present varies enormously, being always of course greater when water is collected from the roofs after a period of dry weather; even when the collecting-area is situated well away from a large town, as the foregoing examples, Table III., which represent stored rain-water in the country districts, and where a considerable amount of water is sometimes stored for use.

A glance at Table III. shows that even these waters are polluted unless considerable care is taken: in this Table are examples of stored rain-water where the reservoirs have not been cleaned out for many years, in the case of 3 for 20 years; but No. 4 is a good water, where the reservoir is regularly cleaned out and considerable care is taken to keep the collecting roofs as free from objectionable pollution as possible.

In Table IV. are given some bacteriological analyses taken of vats in various portions of the town, and serve to show that the amount of pollution varies greatly. In some cases the pollution is enormous and renders the water quite unfit for drinking purposes. The description and approximate capacity of the receptacle is appended.

TABLE III.

Description of storage.	Free ammonia.	Albuminoid amm.	Chlorides.	Hardness.	Phosphates.	Total solids.	Cent. Deposit.
Iron tank	·06	·055	·95	3·5	·20	8	Vegetable fibres. Insect scales.
Open trench ..	·039	·045	1·5	2·5	·57	7	Pollen-granules. Cotton fibres.
Concrete tank	·016	·045	·7	2·5	·10	3·60	*Paramecium aurelia.* Rotifera.
Concrete reservoir	·0014	·01	·4	·8	..	4·0	Nil in 500 c.c.

Expressed as parts per 100,000.

TABLE IV.—*Bacteriological Analyses of Stored Rain-water in the City of Georgetown.*

Description of storage.	Capacity in gallons.	Total organisms per c.c. on Agar at 37° C.	*B. coli* per c.c.	Remarks.	Proprietor.
Iron tank....	4000	440	20	*B. proteus vulgaris.*	Private.
Iron tank....	6000	6,800	1000	*Streptococci.*	Public.
Iron tank....	4000	18,400	1000	*B. dysenteriæ.* *B. violaceus.* *Streptococcus.*	Public.
Iron tank....	800	8,400	1000	....	Public.
Iron tank....	4000	15,400	10	*Streptococci.*	Public.
Iron tank....	7800	2,500,000	1000	....	Public.
Iron tank....	70,000	50	Absent 8 c.c.	Roof area protected from contamination.	Public.
Iron tank....	7800	112	4	....	Public.
Iron tank....	4000	48,000	10	....	Private.
Iron tank....	17,000	998	100	....	Public.
Iron tank....	4000	12,800,000	10	*B. pyocyaneus.* *Staph. alb.*	Public.

TABLE IV. (con.).—*Brick, Concrete, or Stone Tanks and Reservoirs.*

Description.	Capacity.	Total organisms per c.c. on Agar at 37°C.	Fæcal organisms per c.c.	Remarks.	Proprietor.
Brick and Concrete	20,000	170,000	1000	Situate at private house.	Private.
Concrete tank.	25,000	2,080	1	*B. lactis ærogenes.* *B. cloacæ.*	Private.
Brick tank .	20,000	900	1	....	Public.
Brick tank ..	20,000	400	Present 10 c.c. Absent 1 c.c.	*B. pyocyaneus.*	Public.

TABLE V.—*Wooden Vats.*

Construction.	Capacity.	Total organisms per c.c. on Agar at 37°C.	*B. coli* per c.c.	Remarks.	Proprietor.
Wood.......	3000	512	10	......	Private.
Wood.......	5000	2650	10,000	......	Private.
Wood........	2000	984	10	......	Private.
Wood........	3000	1920	1000	Covering very imperfect.	Private.
Wood........	2000	100	1	Well covered, recently cleaned.	Private.
Wood........	2000	1020	100	......	Private.
Wood........	2000	3261	Absent 3 c.c.	Well covered new vat.	Private.
Wood.......	2000	3200	100	......	Private.
Wood.......	2000	330	10	......	Public.
Wood.......	2000	148	1	......	Public.
Wood.......	3000	2,384,000	1000	......	Private.
Wood	2000	2480	100	......	Private.
Wood	2000	30	5	......	Public.

Insurmountable difficulties have been found in working with gelatine plates, so that in all cases agar plates alone have been used. The above analyses can be taken as typical of the general water-supply of the town.

They are taken from a large number of samples without any attempt at classification. The numbers of fæcal organisms vary greatly; it is by no means uncommon for stored rain-water to show the presence of *B. coli* in 1 c.c. only, and later to find *B. coli* present up to 100 per c.c., or even higher, after a shower of rain has washed more polluted material into the vat.

In July 1907 stored rain-water in 6 tanks at the Alms House in the city of Georgetown was examined [*] and reported on as follows :—

No. 2 tank was grossly polluted and tank 4 was a very suspicious water. Tank 6 also contained dysentery bacilli.

It was also remarked that the extent of fæcal contamination corresponds roughly to the extent of roof drained.

TABLE VI.—*The Condition of the Waters at the Alms House.*

Tank No.	Total Bacteria per c.c.	*B. coli* per c.c.	*Streptococci* per c.c.	Special Organisms.
2	1021	(×)10	(×)10	*B. dysenteriæ.*
3	3000	(×)10	0	*B. pyocyaneus.*
4	900	Atypical *B. c. c.*	0	0
5	10,000	(×)10	(×)10	0
6	2600	(×)10	(×)10	*B. dysenteriæ.*
7	2200	(×)10	0	*B. anthracoides.*

Note.—A repetition of the examination of Nos. 2, 5, and 6 on July 31st confirmed the above.

TABLE VII.

Time.	Total Bacteria per c.c.	Total *B. coli* per c.c.	Total *Streptococci* per c.c.	Special Organisms.
3 days before heavy rain....	350	0	0	*B. pyocyaneus.*
Just after heavy rain	1260	(×)10	(×)10	*B. dysenteriæ.* *B. anthracoides.*
1st day after......	1380	(×)10	(×)10	*B. dysenteriæ.*
4th day after	520	(×)10	0	*B. dysenteriæ.* *B. anthracoides.* *B. pyocyaneus.*
9th day after	150	(×)10	0	*B. dysenteriæ.* *B. anthracoides.* *B. pyocyaneus.*
14th day after	43	0	0	0
18th day after	30	0	0	0

[*] By K. S. W. *Note.*—(×) in front of a figure signifies "more than."

Water from a test vat was examined at intervals before and after rainfall, showing the extent of pollution thus caused (Table VII.).

The conclusions drawn from the above were :—

1. Before the rain the water was good.
2. Just after rain a great pollution occurs and the water is totally unfit for consumption.
3. The pollution slowly disappears till from the 14th day onwards the water is again fit to drink.

In a country like British Guiana, where the annual rainfall approximates 100 inches, the necessity for a purification-process for these stored rain-waters is obvious. In very few instances is self-purification allowed to go on sufficiently long to be effective, before a fresh pollution is added.

Analyses of stored waters in the tanks at the Alms House were also carried out as follows :—

TABLE VIII.

Time.	Total Bacteria per c.c.	Total *B. coli* per c.c.	Total *Streptococci* per c.c.	Special Organisms.
Tank No. 2.				
2nd day after heavy rains	800	(×)10	(×)10	*B. anthracoides.* *B. pyocyaneus.* *B. dysenteriæ.*
6th day after rain	450	(×)10	(×)10	*B. anthracoides.*
11th day after rain	15	0	0	*B. anthracoides.*
Tank No. 6.				
2nd day after rain	10,000	(×)10	(×)10	*B. pyocyaneus.*
6th day after rain	500	(×)2	0	*B. pyocyaneus.*
11th day after rain	110	0	0	*B. pyocyaneus.*
Tank No. 7.				
2nd day after rain	250	(×)2	0	*B. anthracoides.*
6th day after rain	90	0	0	*B. anthracoides.*
11th day after rain	10	0	0	*B. anthracoides.*

Here the tanks appeared to recover from pollution in about 10 days. The organisms, such as *B. pyocyaneus*, *B. dysenteriæ*, &c., were isolated and thoroughly worked out for identification, and their pathogenicity demonstrated on laboratory animals.

A minute from Professor Harrison, on 22. vii. 07, to the Poor Law Commissioners, suggested the Carrion-Crows as being the offenders. With this suggestion the bacteriologist concurred, and with the object of obtaining reasonable proof, the following analyses were carried out on the fæces of the birds, kindly provided by Major De Rinzy as required:—

TABLE IX.—*Carrion-Crow Fæces.*

No.	Total Bacteria per gramme.	Total *B. coli* per gramme.	Total *Streptococci* per gramme.	Special Organisms.
1......	10,000,000	10,000	—	*B. anthracoides.*
2......	10,000,000	100,000	—	—
3......	1,500,000	100,000	—	—
4......	10,000,000	100,000	—	—
5	200,000	10,000	—	*B. pyocyaneus.*
6......	10,000,000	100,000	100,000	—
7......	10,000,000	1,000	—	*B. anthracoides.*
8......	10,000,000	1,000,000	100,000	*B. dysenteriæ.*
9......	10,000,000	1,000,000	100,000	*B. dysenteriæ.*
10......	10,000,000	1,000,000	10,000	*B. dysenteriæ.*

It is obvious from the above that the Carrion-Crows are fully endowed with polluting powers, and from their habits and situation freely exercise them.

In a city of 60,000 inhabitants, with an organised sanitary department, Carrion-Crows are not only superfluous and anomalous, but are dangerous.

On July 1911 an outbreak of diarrhœa was investigated at the Alms House*.

A series of fæces from inmates was first examined as a preliminary to the water-investigation. Of this number examined, 33 per cent. showed the presence of *B. dysenteriæ*, and 20 per cent. the presence of *B. pyocyaneus*. This was of importance, as one of the vats was suspected of being liable to sewage-contamination owing to a defect in its construction. The result of the examination of the water-supplies was as follows:—

* By E. P. M.

TABLE X.

No. of Vat.	Construction.	Total Bacteria per c.c.	*B. coli communis* per c.c.	Special Organisms present.	Degree of Pollution as estimated by amount of P_2O_5 present.
1......	Iron.	184,000	1,000	*B. dysenteriæ.* *Streptococcus fæcalis.* *B. violaceus.*	Heavily polluted.
2......	Iron.	12,800,000	10	*B. pyocyaneus.*	Heavily.
3......	Iron.	280	10	Atypical.	Very heavily.
4......	Iron.	Overgrown plate	10	Atypical.	Slightly
5......	Brick.	900	Present 1 c.c.	Atypical.	Very slightly.
6......	Brick.	400	Absent 1 c.c. Present 10 c.c.	*B. pyocyaneus.*	Slightly.

The *B. coli* isolated were identified by the "Flaginac" test and proved to be typical, and the *B. dysenteriæ* and *B. pyocyaneus* proved to be pathogenic to animals.

The above tables provide ample evidence of pollution of the rain-water supplies of this Colony, and while long residence and frequent use no doubt lead to a certain amount of individual resistance, the ever-present danger is always ready to assert itself.

It has not been possible to obtain any analytical figures dealing with rain-water supplies in other parts of the Tropics—in fact, wherever possible, this mode of supply has been abandoned. Of course, the collection of rain-water for a domestic supply is as old as the history of humanity; it is, indeed, Nature's own process, since in ultimate analysis all rivers, springs, and wells are rain-water. Nature, however, interposes one of two great protections, viz., a large volume of water in the case of rivers and lakes, or a long tortuous filtration through the ground in the case of springs and wells.

The various races of mankind have, however, collected and conserved rain-water regardless of such important requisites: on dirty roofs the rain is collected, in small cisterns the water is kept, and in general filtration is neglected.

The Romans in many places constructed cisterns under the roof into which the rain was led. From these cisterns pipes led the water to all parts of the premises.

In many parts of Constantinople and Bombay such a system is still to be seen. In Calcutta at one time large movable earthenware vessels were similarly used. In Buenos Ayres till recently the roof-water used to be run off into underground cisterns. In many parts of South Africa iron tanks are erected alongside the houses. In the above-mentioned places the original primitive rain-water supply has been replaced by a modern system, including a collecting-area, an impounding lake, settling reservoirs, and filtration with a pipe distribution.

In British Honduras the wooden vats and roof-collection system is identical with that in use here.

In Venice large underground cisterns are constructed of puddled clay, bricked round. In the centre is constructed a well with holes only at the bottom. These cisterns are half-filled with sand, so that any water pumped out of the well has been compelled to filter through the sand.

In Jerusalem drinking-water supplies are found similar to that of Venice.

In Gibraltar there are wells supplying good quantities of water—unfortunately, however, of a brackish quality. This water is used for flushing drains, sewers, &c., and other sanitary purposes. The drinking-water till 1898 was provided by roof-collected water supplemented by condensed water. Regulations were in force for the cleansing of the roofs once a year.

In 1898 a special 14-acre collecting-ground was constructed, enlarged in 1906 to 40 acres, with a scheme for a 17,000,000 gallons reservoir. Between the collecting-area and the reservoirs are interposed a rough rock filter and a polarite filter.

In most towns the rain-water supply is supplemented by shallow wells of a dangerous character, and water brought in aqueducts or in pipes.

In Georgetown, British Guiana, wells have frequently been sunk and are found to be very brackish, frequently highly ferruginous, and unfit as a drinking supply.

The river-water mixes freely with the sea and does not lose the saline character for some 20 to 30 miles up-river. At this point the river-water is brown in colour, contains large quantities of organic materials, and is similar to the pipe-water of Georgetown mentioned earlier in this paper.

The wide adoption of the rain-water system in British Guiana has led to experiments being made to sterilise the water as it lies in the vats. It is fully recognised that the adoption of such a method does not convert this inefficient system into a suitable water-supply, but it is hoped that could the people be induced to adopt this method the evils of a polluted water-supply could be at least lessened, if not avoided.

After careful experiments with various sterilising agents we have finally decided on chlorine as the cheapest and most efficient. The method adopted is simply to add the chlorine in the form of chlorinated lime to the stored water in the form of an emulsion in a small quantity of water.

We use one ounce of fresh chlorinated lime to 1000 gallons of water ; this gives roughly 6 parts per million, containing 2 parts per million of free or available chlorine. The process is most efficient, cheap, and easily worked—three essentials necessary in order to induce people to use it.

It will also deal with successive additions of polluted water up to a certain point, as the 2 parts per million of free chlorine leaves roughly 1·4 parts as a margin of safety over Thresh's limit of ·6.

This is well seen by reference to Table XI. showing a number of investigations taken from stored rain-water supplies purified by this process and taken after varying intervals.

As is shown by reference to No. 10, 11, 12, and 13, the amount of chlorine added, although sufficient to sterilise the water when first added, is not sufficient to deal with successive additions of fresh pollution ; and the chlorine content would seem to rapidly fall below the ·6 parts per 1,000,000 given by Thresh as the lowest limit of safety.

TABLE XI.—*Chlorinated Lime* 1 *oz. per* 1000 *gallons.*

No.	Description of vat.	Capacity, gallons.	Before Treatment.		After Treatment.		Time after treatment.
			Total organisms per c.c.	*B. C. C.* per c.c.	Total organisms per c.c.	*B. C. C.* per c.c.	
1.	Wooden vat	3000	170,000	1000	Nil.	Absent 6 c.c.	24 hrs.
2.	Iron vat	7800	12,800,000	10	50	Absent 6 c.c.	24 „
3.	Iron vat	8000	2,500,000	1000	120	Absent 10 c.c.	4 „
4.	Wooden vat	5000	330	10	14	1 per c.c.	1 hr.
5.	Wooden vat ...	2000	148	Present in 1 c.c.	14	Absent 10 c.c.	2 hrs.
6.	Iron vat	7000	112	4 per c.c.	14	1 per c.c.	1 hr.
7.	Wooden vat	2000	140	10	10	Absent 12 c.c.	5 hrs.
8.	Wooden vat	3000	1920	1000	28	Absent 5 c.c.	2 „
9.	Iron vat	8000	984	10	2	Absent 5 c.c.	2 „
10.	Iron vat	50,000	12,780,000	10,000	998	100 per c.c. 1 oz. $CaCl_2O$ per 10,000 gallons.	12 „
	„	„	„	10,000 (1 oz. per	39 1000gals)	Absent 15 c.c.	6 „
	„	„	„	10,000	142	3 per c.c.	9 days.
	„	„	„	10,000	250,000	10 per c.c.	16 „
11.	Wooden vat	5000	(3200)	100	36	Absent 20 c.c	4 hrs.
	„	„	„	100	20,000	100	9 days.
12.	Wooden vat (*a*) .	5000	2480	100	800	Present 20 c.c. Absent 10c.c.	24 hrs.
	„ (*b*) .	3000	512	10	40	Absent 5 c.c.	5 „
	„ (*c*) .	„	3,384,000	1000	40	Absent 10 c.c.	½ hr.
	„ (*b*) .	..	„	1000	40	Absent 5 c.c. Present 10c.c.	24 hrs.
	„ (*c*) .	..	„	1000	800	Absent 8 c.c. Present 10c.c.	24 „
13.	Wooden vat (*a*) .	2000	2060	100	..	Absent 3 c.c. Present 5 c.c.	4 „
	„ (*b*) .	„	„	100	..	Absent 5 c.c. Present 10c.c.	24 „
	„ (*c*) .	„	„	100	..	per 10 c.c.	14 days.
14.	Wooden vat	2000	....	10	..	Absent 5 c.c.	4 hrs.
15.	Wooden vat ..	5000	6800	1000	30	Absent 10 c.c.	24 ..

We therefore suggest that the chlorination be carried out at regular intervals, and propose monthly treatments at least in the rainy season.

We would commend the process to those who are dependent on stored rain-water.

The cost is very small; the only objection we have noticed is a slight taste of chlorine for the first 24 hours after treatment. This, however, soon passes off, and in the meantime can be got rid of by aerating the water required for drinking by pouring rapidly from one vessel to another before use.

We beg to tender our thanks to Professor Harrison, of Demerara, and to Dr. J. C. Thresh, M.O.H., of Essex, for kind advice and assistance in dealing with the water-supply in Georgetown.

The Prevention and Cure of Beri-beri.

[Fraser & Stanton.—Lancet, Oct. 12th, 1912.]

These authors, whose valuable work on the etiology of beri-beri is so well known, contribute an interesting article dealing with the application of their results to the prevention and cure of beri-beri.

In the first half of the communication they deal with the prevention of the disease, whilst the second half is devoted to the consideration of possible means to adopt for the cure of patients suffering from the disease.

Dealing first with prevention, they point out that it has been clearly demonstrated that rice is rendered harmful by the milling and polishing process to which it is subjected in the preparation of polished rice. In this process there is removed from the grain some substance of high physiological importance, the absence of which results in the production of polyneuritis in fowls and of beri-beri in man when a diet is consumed of which polished rice is the staple.

Although harmless and harmful rices can readily be distinguished by simple inspection, the definition of polished and unpolished rices on the basis of histological differences alone is not sufficient for practical purposes, and it is therefore necessary to have in addition a chemical standard.

For the establishment of such it is necessary to consider the various constituents of the rice-grain and to select some substance which from the relative ease and accuracy with which it could be estimated would serve as an indicator.

It is pointed out that neither the fats nor the protein in the grain are suitable for estimation, as, in the first place, both substances are difficult of extraction, and further that the differences in the amount of these substances when extracted from polished and unpolished grains respectively are so slight as to be of little use.

Attention was therefore directed to the inorganic salts in the grain, and the authors found that of these phosphorus pentoxide was relatively easy of estimation, and, further, that the amount of this salt in the two types of grain showed a marked difference.

They found that with unpolished and therefore safe rice the amount of this salt invariably exceeded 0·4 per cent., whilst in polished or harmful rices the figure for phosphorus pentoxide was invariably considerably less than 0·4 per cent.

The authors suggest, therefore, that this figure of 0·4 per cent. of phosphorus pentoxide should be used as a standard and that any rice whose phosphorus content falls below this figure should be rejected as extremely likely to produce beri-beri.

In recommending the adoption of phosphorus estimations for purposes of standardisation the authors emphasise the fact that they do not thereby subscribe to the view that a deficiency of phosphorus in organic combination explains the production of beri-beri by the consumption of polished rice.

Reference is made to the work of Vedder and Chamberlain, who advocated the estimation of potassium as suitable for standardisation purposes. Drs. Stanton and Fraser have not employed this method, but see no reason why it should not serve the purpose of an indicator quite as well as phosphorus pentoxide.

The vexed question of legislative measures to prevent the use of polished rice is reviewed and the various measures suggested and the great difficulties of the problem are discussed.

A note of warning as to the dangers of hasty and ill-considered legislation on the matter is sounded by the authors when they state :—"Undue haste in the application of the results of scientific research to practical and actual conditions has so often in the past been attended by unsatisfactory and even disastrous results that anything in the nature of sumptuary legislation should be introduced only after grave consideration. It has been proposed to make the use of unpolished rice compulsory by legislation. With this object in view, it has been suggested that the importation of polished rice should be prohibited, but despotic legislation of this kind would be dangerous, impolitic, and in certain places a menace to trade."

In considering the problem of the cure of beri-beri the well-established fact that the administration of polished rice together with the polishings of that rice did not produce beri-beri was used as a starting-point of experiments to produce some substance from these polishings which could be used as a curative agent.

As collected in rice-mills, polishings are mixed with dust and other adventitious substances. Such a mixture, either as an emulsion or as a gruel, does not look very palatable, and it is estimated that an adult accustomed to the use of polished rice would require not less than 1·75 ounces of polishings daily; in this form there would be involved the administration of a quantity of substances unnecessary and perhaps undesirable for a sufferer from beri-beri.

Means were sought therefore to separate the active substances from the mass of polishings and so prepare an agent which could be given in a readily available form and free from useless and undesirable material.

Previous experiments had shown that the active substances in the polishings were soluble in water and in 91 per cent. alcohol, and by carefully devised chemical processes the authors were able to make a solution of these active substances in a mixture of water and alcohol.

The next step undertaken was to treat fowls who had been exclusively fed on polished rice, which had produced in them a well-marked polyneuritis, with this extract of the polishings to ascertain if it possessed any curative effect.

Six experiments were undertaken, and in five of them complete recovery ensued in some 32/40 days.

A further test was carried out to test the prophylactic value of the extract.

Eight fowls were fed on polished rice and received daily 0·5 c.c. of the extract. The experiment extended over five weeks. The fowls were in perfect health at the conclusion of the period and their weights throughout were equally satisfactory.

Still further experiments were undertaken, aimed at the more exhaustive extraction of the active substances of the polishings by treating them with hot acidulated alcohol.

Six cases of polyneuritis in fowls were treated with this second extract, of which each case was given 1 c.c. daily. The fowls continued to receive the polished rice on which they had developed the disease. In all six cases complete cure ensued in some 34/48 days.

So far the investigators have not been able, owing to various difficulties, to produce these extracts in large quantity suitable for administration to a series of human cases, but it is hoped shortly to overcome these difficulties. It is then proposed to ascertain if these extracts exert any curative effect on man.

The results of such administration will be awaited with the greatest interest.

H. B. N.

VI.—CLINICAL MEDICINE.

CASES OF TRYPANOSOMIASIS IN ENGLAND, MAINLY AT THE LONDON SCHOOL OF TROPICAL MEDICINE. (2nd Report.)

BY C. W. DANIELS, M.B., M.R.C.P.

IN the first number of this Journal I recorded the cases of Trypanosomiasis that had been under observation in England at the London School of Tropical Medicine up to October 1911. Of the 17 cases there recorded, 5 had died and 12 were then living. In this paper I propose to give an account of the further progress of these 12 cases as far as is known, and of two additional ones admitted to the Albert Dock Hospital in 1912. Eleven out of the 12 are known to be still living, but of one (*Case* 10, E. G.) we have had no further information. Of the eleven, one thus survives 11 or 12 years from the onset of the symptoms, one 10 years or more, three over 7 years, two 6 years or more. As these cases appear to be in good health, have no symptoms of trypanosomiasis, and no trypanosomes can be found in the blood, probably all 7 may be considered as recoveries.

Of the other four who have survived from 2¾ to 5½ years, three are free from any symptoms, and repeated examinations of the blood have failed to show any trypanosomes: these are probably on the way to recovery and are engaged in the active pursuit of their duties. One of them (*Case* 17, C. W. B.), at the date of the last report, had been treated only by intravenous injections of tartar emetic, under which treatment he improved, but trypanosomes recurred after the injections were discontinued. In October last year another series of injections were given, but as trypanosomes again recurred he was put on a course of intramuscular injections of atoxyl, 3 grains twice a week. No further recurrence has taken place, and he has returned to duty in West Africa. At no time were his parasites either antimony resistant or atoxyl resistant, but the long-continued treatment with atoxyl seemed to be required to supplement the action of tartar emetic, which alone had not a sufficiently permanent effect.

In this case the prognosis appears to be distinctly good, but as the onset of the symptoms was in July 1910, 2 years and 2 months ago, and trypanosomes were last found about 9 months ago, the period is too short to as yet claim this case as a recovery.

The remaining case, No. 16, W. G., has been under treatment since October 1909, about a fortnight after the first onset of the symptoms, and he is one of the three cases from Rhodesia we have had in our hospital. The progress up to October 1911 has been fully recorded, and since then there has been little change. Trypanosomes are still occasionally present; there are attacks of pyrexia lasting for one or two days every 8–12 days, and occasionally there are glandular swellings. The general health continues good, and the severer symptoms which he had two years ago are absent. Intravenous injections have had to be abandoned, as there are no superficial veins remaining of sufficient size. Antimony has been given by rectum, but probably little is absorbed, though the occasional occurrence of physiological symptoms renders it probable that it is in part. Atoxyl is given regularly.

There is no doubt that this is a typical Rhodesian case, and to a large extent atoxyl resistant. The peculiar morphological forms found in rats inoculated were not to be found for many months, and afterwards were only found with difficulty after passing through several rats. Now they are found more frequently, but only in small numbers; fuller details are contained in Wenyon's paper. He calls the strain G.

Case 18. H. R. T., aged 25, Southern Rhodesia, since August 1908.—Beyond slight attacks of malaria he had good health till March 1912. The disease commenced with fever and vomiting followed by severe epistaxis lasting for 12 hours. The severer symptoms subsided, but fever continued and he had insomnia and at times delirium. He also had visual disturbance and was unable to see out of the left eye—? keratitis or iritis. He rapidly lost flesh and became anæmic, with general muscular weakness. He was admitted to the Albert Dock Hospital on July 31, trypanosomes having been found two days previously. On admission he was thin and wasted, markedly anæmic, and muscularly weak. Red corpuscles were 3,100,000. The temperature varied from 100°–102° F. There was no erythematous rash. The liver and spleen were not enlarged. There was marked delayed hyperæsthesia in the muscles of the leg. The left testicle was markedly enlarged. No enlarged glands were detected.

The patient was given atoxyl, 3 grains on alternate days, by intramuscular injections, and rectal injections of 10 grains of tartar emetic. The temperature gradually fell, rarely passing over 100° F., and falling frequently to normal or subnormal. Trypanosomes were found, 1 to 12 per 200 leucocytes, and sometimes none were found. An enlarged gland behind the sternomastoid appeared on Aug. 12th. On Aug. 19th he had some pain in the throat and difficulty in swallowing; this was followed by hoarseness and aphonia and free discharge of copious frothy sputum after the 22nd. On Aug. 26th he sat up suddenly in bed and dropped back dead.

Notes.—This patient had never been north of the Zambesi. The station on which he was working was a small one 300 miles north of Buluwayo. There was no Medical Officer, and no cases of sleeping sickness were known in the neighbourhood. The patient did a good deal of shooting, and *Glossina morsitans* was common, but *Glossina palpalis* was unknown. Probable duration of the disease under 6 months. The absence of a rash, the late appearance of enlarged glands, the testicular swelling, and death without pronounced nerve-symptoms are noteworthy.

Many of the aberrant forms of trypanosomes up to 40 per cent. of the total are to be found in inoculated rats. Wenyon describes the strain as T.

A. A. E., 28.—This case was detected and the earlier stages have been recorded by Dr. Stannus, of Nyasaland. The disease commenced about April 4th. The temperature was continuously high, and there was rapid emaciation and great weakness. He was treated in Zomba with a few intravenous injections of tartar emetic, and arrived in England towards the end of July, and was admitted to the Albert Dock Hospital on Aug. 1. Very sallow and anæmic, limbs much wasted, and spleen greatly enlarged. There was marked tenderness on pressure on the muscles of the legs, more marked than in any previous case. No erythematous eruption. Glands in groins, axilla, and neck readily palpable, not markedly enlarged. The testicles were swollen early in the illness, but were not enlarged on admission. Patient was put on atoxyl and rectal injections of tartar emetic, and at first improved. The temperature was lower, and the trypanosomes, which were during the first 4 days in dry films in the proportion of 10–23 per 200 leucocytes, were so much reduced that on some days none were found and on others only one or two. On Aug. 24 the temperature rose higher, 101°·6, and the trypanosomes increased to 33 per 200 leucocytes, and on the following day were more numerous than the leucocytes, 250 to 200 leucocytes. The patient appeared to be very ill, there was œdema of the abdominal walls, and on Aug. 26 1½ grains of tartar emetic were given intravenously, and this was repeated on Aug. 30 and Sept. 3, 7, 10. The general condition improved, trypanosomes in the blood became scanty, and the muscular tenderness disappeared. He left the hospital at his own desire, and died suddenly on Sept. 30. Probable duration of the disease 6 months.

In this case also there had been no definite erythema of the characteristic type. The glandular enlargement was slight, the testicular enlargement transient, and the splenic enlargement, the muscular tenderness, and the anæmia very marked.

The disease was contracted in Portuguese East Africa. Other Europeans who had been living in the same house had not contracted the disease. It was not known to exist amongst the natives, and though *Glossina morsitans* was common, *Glossina palpalis* was not known. The patient was a professional big-game hunter.

The trypanosomes in rats inoculated from this case included a proportion of forms with the macronucleus posterior. The proportion was much smaller than in strain T, but larger than in strain G. Wenyon in his preliminary note describes it as strain E.

These three cases are all undoubtedly due to the variety (? species) described as *Trypanosoma rhodesiense*. They are all of greater severity and more resistant to treatment than the cases we have had from Uganda and West Africa. Clinical variations have been common in these cases, but are more so in the cases from Rhodesia. In two out of the three there was orchitis and in only one of the other cases. Anæmia was very marked in two and marked in the third, W. G.; whilst in many of the other cases it was slight or not present. Delayed muscular pain on trivial injuries was specially marked in the Rhodesian cases, and in one, E, the muscles were extremely tender, and pain was caused on any attempt to contract the muscles.

Although the symptoms were more severe and complications more numerous, there were no symptoms that have not been observed in cases from other parts of Africa.

As regards the trypanosomes themselves, they were more numerous than in the other cases, but in man were indistinguishable from *T. gambiense*. In rats the aberrant (? involution) forms occurred in all, but in varying proportions.

The malignancy of the parasites on the lower animals is greater than in trypanosomes isolated from other parts of Africa including Katanga.

In our three strains the specific character is now present in all, but (*vide* Wenyon's paper) the aberrant forms vary in their proportion, and the malignancy of the strains varies.

Treated with Liq. arsenicalis and other drugs.

Case.	Initials.	Probable date of onset of symptoms.	Trypanosomes found.	Latest reports as to signs of the disease.	General Result.	Duration of disease n years.
1	H. K. (Ford & Dutton's Case).	. . . .	. . . .	. . . .	Death Jan. 1903—Cerebral symptoms.	$1\frac{2}{3}$
2	Mrs. M.	1900 1901	July 1902	None	Good health.	11–12
3	Mrs. S.	1901	Oct. 1902	. . . .	Death Nov. 1903—Sleeping Sickness.	$2\frac{1}{4}$
4 . .	J. M.	June 1902	March 1903	. . . .	Death 1906—Sleeping Sickness.	4
5	Mrs. G.	?	1902	None	Good health.	10 or more
Treated with Atoxyl.						
6 . .	Z.	?	July 1905	None	Good health.	over 7
7	Mrs. R.	June 1905	Sept. 1905	None	Fair health.	$7\frac{1}{3}$
8	H. C. C. S.	1905?	Feb. 1906	None	Good health.	about 7
9	W. R. E.	Sept. 1906	Jan. 1907	. . . .	Death March 1908—Pneumonia.	$1\frac{1}{2}$
10	E. G.	Dec. 1907	Jan. 1908	None	Good health.	5 or more?
Treated with Atoxyl and Antimony either by mouth or by injection.						
11 . .	C. G.	Nov. 1906	Jan. 1907	None	Good health.	6
12 . .	C. V.	July 1909	June 1910	None	Good health.	$3\frac{1}{4}$
13 .	A. P.	July 1907	July 1907	None	Good health.	$5\frac{1}{4}$
14 . .	D. H. L.	Aug. 1908	Aug. 1909	. . . .	Death 1911—Septicæmia.	$2\frac{10}{12}$
15 .	Dr. McD.	Dec. 1909	Dec. 1909	None	Good health.	$2\frac{3}{4}$
16 . . .	W. G.	Sept. 1909	Oct. 1909	Present	Fair health.	$3\frac{1}{12}$, still infected
17	C. W. B.	July 1910	June 1911	None	Good health.	$2\frac{1}{4}$
18	H. R. T.	March 1912	July 29, 1912	. . . .	Died suddenly Aug 26, 1912.	$\frac{5}{12}$
19	A. C. E.	April 1912	May 1912	. . . .	Died suddenly Sept. 30, 1912.	$\frac{5}{12}$

The very important practical questions arise as to

(1) Is the parasite a different species?

(2) Is the parasite a more virulent strain of the same species modified by passing through a different cold-blooded host, *Glossina morsitans*, and perhaps by passage through warm-blooded hosts other than man?

Or (3) Is it a species in course of development?

Much further work is required before a definite answer can be given to these questions, but it is not a matter of purely theoretical importance.

As regards the first. If a new species, from where has it been derived? The forms described are not found in the strains derived from cases from the Congo, from West Africa, from Uganda, or even from Katanga, south-west of Tanganyika—all places where now the infection is from man to man through *Glossina palpalis*. The "species" must be indigenous, either in man or animals, in Rhodesia and the surrounding districts or imported from places at no great distance.

(2) The clinical differences in the cases are of the same character as those which are used to differentiate the different species of blood-spirochætæ, but the variability in the characteristics of the different trypanosomes isolated from the different cases are more such as we expect in different strains rather than in well-defined species.

(3) Whether, assuming it to be merely a strain modified according to the host, the modifications will become so definite and uniform as to elevate it to specific rank time only can show.

The steady extension of trypanosomiasis south is a very serious matter. The expeditions in Rhodesia have proved conclusively that *Glossina morsitans* is a carrier. That the important reservoir is the African wild game derives some support from the fact that these three cases were all in game-shooters, two of them professional ones, and that there was no evidence of human infections in the vicinity. Now that a case in a European has been detected after arrival in England does not indicate that this was the only infection south of the Zambesi, and further extensions south are to be feared. Expeditions have done their work, and it is to be hoped that well-organised energetic measures, even if these involve the extermination of the wild game in broad belts, will be taken, commencing in the districts known to be free from infection and steadily working towards the known infected districts. Centripetal preventive measures must replace the centrifugal observations of the spreading area of infection, which so far have been an object of the expeditions, and an organised Resident Medical Service will be required with one or more central laboratories, instead of expeditions which, though their results have been of great value, have served their turn. Steady work by Resident Medical Officers will now give the best chances of checking or stopping the spread of the disease into the districts further south where *Glossina morsitans* occurs.

REPORT ON TREATMENT OF TWENTY-TWO CASES OF YAWS BY SALVARSAN.

[R. P. COCKIN.—Journal of Tropical Medicine and Hygiene, Sept. 16th, 1912.]

SINCE the introduction of Salvarsan some time back, numerous observers, in addition to employing it in the treatment of syphilis, have tried it as a remedy in other diseases with varying success.

Evidence is now accumulating which seems to show its marked efficacy in cases of yaws, a disease which hitherto has shown marked resistance to practically every form of treatment adopted.

An interesting report is furnished by Cockin showing the results of treatment with salvarsan of 22 cases of the disease at Grenada, West Indies.

The patients were of both sexes and of ages varying from 3 years to 37 years.

Injections of the drug were in all cases made intra-muscularly, the drug being prepared for injection according to the method of Tæge with slight modifications.

The dosage employed for adults was 0·6 grm., whilst for children 0·2 or 0·3 grm. was the amount usually administered.

The results recorded in all cases are remarkable, all evidence of the disease disappearing rapidly and the patients being discharged cured usually within a month from the date of injection.

Two of the cases recorded are of especial interest. Both had been under treatment with various other remedies for 18 months with practically no benefit. Under salvarsan, both cases improved rapidly and were discharged cured seventeen and twenty-nine days respectively after injections.

Only one case of the series required a second injection to effect the cure, and in this case it appeared as if the primary injection 0·3 grm. was too small. No progress towards recovery was made until a second injection of 0·4 grm. was given, when the disease rapidly yielded.

In no case did abscess at the site of injection result, nor was the pain produced by injection at all severe.

Two of the cases treated, in children of 6 years and 7 years respectively, showed somewhat marked symptoms of arsenical poisoning and occasioned a good deal of anxiety, but eventually recovered and were completely cured of the disease for which they were treated.

No definite cause for these alarming symptoms could be made out, and Cockin considers that they must be ascribed to some peculiar intolerance of the drug present in these children, as another child of three years of age, who was injected on the same day with the same dose from the same tube of salvarsan, and with the same syringe and needle, presented no untoward symptoms whatever.

The results of treatment of the whole 22 cases are presented in a neat tabular form, which shows at a glance the remarkable results obtained.

H. B. N.

ABSTRACT FROM THE 'BRITISH MEDICAL JOURNAL,' Aug. 24th, 1912.

FURTHER EXPERIENCE OF THE SPECIFIC CURATIVE ACTION IN AMŒBIC DISEASE OF HYPODERMIC INJECTIONS OF SOLUBLE SALTS OF EMETINE.

By LEONARD ROGERS, M.D., F.R.C.P., I.M.S.,
PROFESSOR OF PATHOLOGY, CALCUTTA.

IN the 'British Medical Journal' of June 22nd the author recorded cases illustrative of acute and chronic amœbic dysentery and acute hepatitis respectively, in patients who were unable to take ipecacuanha by the mouth, but were rapidly cured by subcutaneous injections of soluble salts of emetine, without the production of vomiting or marked nausea, which are such serious drawbacks to the use of full doses of ipecacuanha by the mouth. During the two months which have elapsed, the author has had frequent opportunities of using the new treatment in suitable cases. The results leave no possible doubt as to the great practical importance of the discovery: some of them, indeed, so surprising that they must be seen to be fully appreciated, as words can convey but a feeble impression of the rapidity and completeness of the recoveries effected in very grave cases.

The cases comprising the more extensive material now available are classified into different clinical groups:—

1. *Extremely acute amœbic dysentery, with marked thickening and tenderness of the bowel and high leucocytosis.*—The prognosis in these cases is exceedingly grave—gangrene, perforative peritonitis, or post-colic abscess are frequent fatal complications. Of thirteen such cases with leucocyte counts of 25,000 to 49,250, eleven were fatal within a few days and only two recovered. Ipecacuanha had been given in quantities of 30 to 60 grains or more a day. The fact is, sufficient ipecacuanha cannot be administered quickly enough by mouth to save more than a very small percentage of such cases. Details are given of two cases of the acutest form of amœbic dysentery in which the disease was cut short, and both great thickening of the bowel and an extreme degree of leucocytosis cleared up within three or four days, very great improvement having been apparent within 24 to 48 hours, under treatment with from 1 to 1½ grains of emetine hydrobromide daily, an equivalent of 90 to 135 grains of ipecacuanha.

The next case (3), one of gangrenous amœbic dysentery, proved fatal. Careful post-mortem examination revealed the fact that all amœbae had been destroyed within sixty hours by emetine injections, totalling 3½ grains. The failure to save this opium-eating patient furnished the most conclusive evidence of the specific nature of the new method of treatment, and fully explained the apparently miraculous recoveries recorded in cases 1 and 2.

2. *Moderately acute amœbic dysentery rapidly cured by emetine.*—Details are given of two cases (4 and 5) of typical acute amœbic dysentery such as usually yield to full doses of ipecacuanha in time, but which are much more rapidly and certainly cured by the new treatment and with far less suffering to the patient.

3. *Chronic amœbic dysentery rapidly cured by emetine.*—Case 6, amœbic dysentery of six months' duration. Twenty-four hours after the first injection

of ½ grain of emetine, stools were free from blood or mucus and no amœbæ could be found. The ½ grain injection once a day was given for three days, after which ipecacuanha 20 grains was given every evening.

Another patient with dysentery of 3½ months' duration, recorded in the author's first paper as rapidly cured by emetine, has continued free from all dysenteric symptoms.

4. *Acute hepatitis threatening liver-abscess formation, cured by emetine.*—The remarkable effect of ipecacuanha in preventing liver-abscess by curing both acute and chronic hepatitis, demonstrated by the author in 1907, has been repeatedly confirmed by others since. In a first paper on the emetine treatment was recorded an example of the rapid action of the new method in a patient who could not take ipecacuanha by mouth. Two further cases (7 and 8) are detailed, illustrating the value of emetine.

5. *Amœbic abscesses of the liver and spleen cured by aspiration and emetine injections.*—In 1902 the author showed that unopened amœbic abscesses of the liver are, in the majority of cases, sterile as regards bacteria, and therefore suggested removing the pus by aspiration and injecting soluble salts of quinine into the cavity to kill the amœbæ in the wall of the abscess, in place of the exhausting open operation with prolonged drainage. Later, the author advised, in addition, the continued administration of full doses of ipecacuanha after any form of operation on such abscesses. The method is being used with increasing success in Calcutta at the present time, though it fails in some cases. The proof afforded in the author's recent paper, that soluble salts of emetine rapidly kill the amœba of dysentery when used in much higher dilutions than the quinine salts, clearly indicates the former drug as the more efficient one for the purpose just mentioned. Moreover, the evidence above recorded in this paper, to show that emetine can be given in sufficient doses to kill off rapidly all the amœbæ in the greatly thickened wall of the large bowel in acute dysentery, made it highly probable that it would act equally well on the parasites in the walls of amœbic abscess of the liver. Details of a case (9) are given, showing that this actually happens. The case was one of acute amœbic dysentery with multiple liver-abscesses, in which all the amœbæ in the latter were killed by subcutaneous injections of emetine in ½ grain doses twice on two consecutive days. The careful post-mortem investigation and microscopic examination left no doubt that every amœba in the liver had been killed by the emetine injected subcutaneously during life, as the body was fresh and the temperature very favourable for the preservation of the parasites at the time of autopsy. This case, taken with No. 3 above recorded, affords strong evidence that emetine salts administered hypodermically in sufficient doses will kill all the amœbæ in both the intestinal and liver-abscess walls. Further cases detailed are :—

Case 10.—Epigastric liver-abscess cured by aspiration and emetine injections after failure of the quinine treatment. At the second aspiration 1 grain of emetine hydrobromide dissolved in 2 oz. sterile salt solution was injected into the cavity ; the emetine salt was also injected subcutaneously in ¼ grain doses every morning for four days, and again on the sixth day. In addition, 10 grains of ipecacuanha were given by mouth every evening.

Case 11.—Right lobe liver-abscess cured by aspiration and injection of emetine. Here, again, emetime was administered hypodermically in ½ grain doses, and 1 grain of the hydrobromide in 2 oz. sterile salt solution injected into abscess-cavity at time of aspiration. Ipecacuanha by mouth was given also.

A third case, very similar to No. 11, has done equally well, but it is yet too early to be certain of his permanent cure. In two others, the pus obtained at the time of aspiration and injection of emetine was found on culture to contain large numbers of staphylococci, and consequently the abscesses had to be opened and drained, with ultimate recovery.

Amœbic liver-abscesses cured by aspiration and subcutaneous injection of emetine.—In two other cases, one of which is still under observation, liver-abscess pus was evacuated by aspiration and emetine injected subcutaneously, but not into the abscess-cavity. In one, the emetine injections were given for acute hepatitis, which greatly improved; but as leucocytosis persisted, the liver was explored and several ounces of pus withdrawn, after which steady convalescence ensued and the leucocytosis disappeared. In the other, the emetine injections were commenced two days after the aspiration of liver-pus, with an equally happy result. A single aspiration so exceptionally cures such cases that these two successive recoveries are in all probability due to the action of the subcutaneous injection of emetine in destroying the amœbæ in the walls of the liver-abscesses; so it may eventually prove unnecessary to inject the drug into the cavities.

Notes are given of case 12:—*Amœbic abscess of spleen* cured by aspiration and emetine injections into cavity of abscess and subcutaneously. This case is of interest, both on account of the rarity of amœbic abscess of the spleen and because of its successful treatment by the new method. Such cases when opened heal very slowly and leave obstinate sinuses. In this case recovery was complete and rapid.

The disappearance of leucocytosis as evidence of the cure of an amœbic abscess of the liver.—It is often difficult to decide if the abscess is completely cured, or if some pus has again accumulated, necessitating a further aspiration. Here leucocyte counts are of great value—the author having frequently found that if an original actual or relative leucocytosis has not disappeared within about a fortnight after an aspiration, pus will usually be again found on exploring; while if the original increase in the white corpuscles has vanished, the abscess is really cured and further aspirations are negative, though harmless.

Dosage.—Hydrochloride and hydrobromide of emetine are equally useful, the former being the more valuable. The author at first employed $\frac{1}{8}$ grain doses, but now very seldom gives less than $\frac{1}{2}$ grain at a time, and often as much as $\frac{2}{3}$ of a grain, the equivalent of 80 grains of ipecacuanha; and twice has injected 1 grain subcutaneously without any vomiting or depression, but such a quantity is only required in extremely acute amœbic dysentery. The salts can be safely boiled for a very short time, but it is better to dissolve them in sterile saline, or to boil the solution first and then add the emetine salt.

The value of emetine treatment in the differential diagnosis of amœbic from other varieties of dysentery.—The very great rapidity with which marked improvement follows the subcutaneous injection of $\frac{1}{2}$ grain doses of emetine is of the greatest diagnostic importance, for cases of bacillary dysentery and other non-amœbic causes of the presence of blood and mucus in the stools are not, in the author's present experience, materially affected by the drug, although it has done no harm in them. Two cases are cited, illustrating the diagnostic value of the drug. The author remarks that facilities and time for the microscopic examination of the stools of the large number of dysentery cases which often have to be dealt with in the tropics are seldom available; so that a simple and harmless clinical method of differentiating amœbic from other forms, such as is now furnished by the emetine treatment, is of the greatest practical importance and will also lead to the early adoption of suitable treatment in those cases which prove not to be amœbic in nature. Moreover, it will allow of more rapid advance in our knowledge of the distribution of the two great classes of dysenteries than can be furnished by the very limited number of workers available for bacteriological researches on this subject in the tropics.

H. M. H.

I.—DEPARTMENT OF ENTOMOLOGY.

Remarks on the Systematic Position of *Anopheles sinensis*, Wiedemann.

By Col. A. ALCOCK, C.I.E., F.R.S., I.M.S. (retired), and Miss SOPHIA L. M. SUMMERS, M.A., B.Sc.

The magnification of the genus *Anopheles* to a "subfamily" and the parcelling of the species into a multitude of "genera" have proved so inconvenient to medical men that we think it opportune, though intrinsically the subject is trivial, to show, from examination of a large series of a particular species of *Anopheles*, how unstable are some of the characters which have been used as standards in this elaborate scheme—we cannot call it system—of classification. It may, perhaps, be argued that, like Sir John Falstaff at Shrewsbury, we are slaying the slain, since so many entomologists are already convinced that all the species of the mosquitoes whose larvæ are without a breathing-tube form an indivisible generic group; but we think that no harm is done by furnishing evidence for testing opposite opinions. Our own view is that the genus *Anopheles*, Meigen, exhibits a most beautiful series of slight gradations—a series, perhaps, as nicely graded as any in the zoological system, from forms, like *Anopheles maculipennis*, with sparsely hairy body and uniform colouring, to forms like *Anopheles pulcherrimus* ("*Cellia pulcherrima*"), with scaly body and much diversified colouring.

1. Anopheles sinensis *as a supposed Generic Type.*

The species examined is *Anopheles sinensis*, Wiedemann ("*Myzorhynchus sinensis*"), in two individuals of which, it may be mentioned, Stanton, at Kuala Lumpur, found malaria zygotes. Of this species we have tabulated the characters of 51 females and 8 males from Kuala Lumpur and also of 8 females from India.

Anopheles sinensis, Wiedemann, was proposed by Theobald, in the 'Journal of Tropical Medicine' for 1902, as the type of a genus *Rossia*, characterised as follows:—"Thorax with hair-like scales, abdomen with ventral and apical scales; wing-scales broadly lanceolate; palpi densely scaled in the female, and also the proboscis." The name *Rossia*, being already occupied in the system, was changed by Blanchard to *Myzorhynchus*.

In the Tables of Genera, in the 4th and 5th volumes of Theobald's 'Monograph,' the characters of the genus *Myzorhynchus* are thus summarised:—"Thorax with hair-like curved scales; no lateral abdominal tufts; distinct apical ventral tuft; palpi densely scaly; wings with dense large lanceolate scales." The "apical ventral tuft" is a wisp of scales standing out in the mid-ventral line from the 7th abdominal segment of the female; it is conspicuous in the female of *A. sinensis*, but in the male is either absent or reduced to one or two small scales; and it has commonly been accepted as a distinguishing feature of the "genus *Myzorhynchus*."

The component species of the proposed genus (*vide* 'Monograph of Culicidæ,' vol. v. p. 49) are the following:—*Anopheles barbirostris*, v. d. Wulp; *A. pseudobarbirostris*, Ludlow; *A. bancrofti*, Giles; *A. umbrosus*, Theobald; *A. strachani*, Theobald; *A. albotæniatus*, Theobald; *A. sinensis*, Wiedemann; *A. vanus*, Walker; *A. pseudopictus*, Grassi; *A. minutus*, Theobald; *A. nigerrimus*, Giles; *A. mauritianus*, Grandpré; *A. paludis*, Theobald.

Now, as regards scales on the abdomen, the 13 "species" of the above list fall into three sets:—

In one set, including *A. sinensis* and *A. mauritianus* (= *A. paludis*), the specified tuft is conspicuous in the female, but is absent (or is represented by not more than 2 scales) in the male, and both sexes may also have some scales on the 8th segment and genital lobes.

In another set, including *A. umbrosus* (= *A. strachani*) and *A. albotæniatus*, the female has neither tuft nor scales of any kind on the abdomen.

In a third set, including *A. barbirostris* and *A. bancrofti* (= *A. pseudobarbirostris*), not only is the specified tuft conspicuous in the female and represented feebly in the male, but scales are also present on the ventral surface of every segment behind the 1st in the female and on a varying number of segments in the male. In the female these additional scales usually stand out as a clump on each segment, but occasionally they almost cover the ventral surface.

We may conclude, therefore, that although the species of *Anopheles* named above are closely akin—more closely akin one to another than to a form like *Anopheles pulcherrimus*, for instance,—yet they do not form a group capable of being exactly defined by the distribution of abdominal scales. As a matter of fact, they are links of a chain connecting species like *Anopheles maculipennis*, which has hairs but no true scales on thorax and abdomen, with species like *Anopheles maculipes* ("*Arribalzagia*" *maculipes*), in which with a hairy thorax there are plenty of scales on the abdomen. So that, taking scales into consideration (and the same argument can be shown to hold good for other characters), the alternative is either (*a*) to make every link of the chain a separate genus or (*b*) to include the whole series in one comprehensive generic group. If the former alternative have not even the argument of practical convenience to recommend it—for practical convenience must often be allowed to make short work of the claims of Nature,—then no hesitation need be felt in emphasising its almost grotesque disregard of the aim and object of a classification.

2. Anopheles sinensis *in its Specific Relations.*

Besides distribution of abdominal scales, *Anopheles sinensis* has certain other characters which distinguish it, or are supposed to, from the other closely-related species which have been dubbed "*Myzorhynchus*." From *Anopheles mauritianus* (= *paludis*) it is obviously distinguished by the colouring of the tarsi of the hind legs. From *Anopheles vanus*, *A. pseudopictus*, and *A. minutus* it is supposed to be distinguishable by the presence in the fringe on the posterior edge of the wing of a pale spot, and from *A. nigerrimus*, furthermore, by having the tip of the palpi white.

The value of these supposed specific distinctions may be estimated from the following summary of the results of a very careful examination of 54 females of *A. sinensis* caught or bred, not all at the same time, in one place (Kuala Lumpur) :—

(*a*) As regards *the colouring of the wing-fringe* : At the tip of the wing, where the fringe begins, there is always a yellow blotch, the size of which varies considerably even in the two wings of the same individual. The rest of the fringe is usually dark, with a pale break at the ending of the posterior branch of the 5th longitudinal vein : but this pale break may be visible only when the light falls at a particular angle, or it may be altogether absent ; occasionally the fringe is mottled with pale patches in all its extent.

(*b*) As regards *the colouring of the palpi* : The palpi are black, and commonly there are white scales at or near the far end of every segment, so that they are usually described as black with three white bands and white tip.

The white tip is very constant, occasionally it cannot be distinguished with the naked eye, on one palp only.

The white band at the end of the 3rd segment is the next in constancy, but it may be extremely indistinct, and occasionally it spreads broadly and runs into the white band at the tip of the 4th segment, on one or both palpi.

The white bands at the ends of the 2nd and 1st segments are variable : both of them may be very distinct, or one or both of them may be represented by a few white scales that can hardly be seen by the naked eye.

In addition to the distinct white bands (4, 3, or rarely 2), white scales may be scattered, sometimes quite plentifully, on the 3rd and 4th segments of the palpi.

(*c*) As regards *the colouring of the legs* : The tips of the first three tarsal segments of the front and middle legs, and of the first four of the hind legs, are always light-coloured, so that the tarsi are described as banded. In the hind legs these light-coloured markings range from mere pale spots to broad white bands.

Our observations, therefore, confirm the opinion of those who regard the names *A. vanus*, *A. pseudopictus*, *A. minutus*, *A. nigerrimus*, and also *A. peditæniatus* of Leicester as synonyms of *A. sinensis*, Wiedemann.

We also conclude that "species" of *Anopheles* based upon colour-markings of palpi, wing-fringe, and tarsi—or even upon the minute distribution of scales on the abdomen—are based upon assumptions that require to be verified by examination of a considerable number of individuals.

3. *Conclusion.*

In our view, which we consider is justified by the facts summarised above, the species of *Anopheles* that have been segregated by numerous writers as "*Myzorhynchus*" are only four in number, viz. :—

1. *Anopheles sinensis*, Wiedemann : female with a tuft of scales in the mid-ventral line on the 7th abdominal segment ; palpi and tarsi banded.—Oriental, extending into Palæarctic Region.

2. *Anopheles mauritianus*, Grandpré : female with a tuft of scales in the mid-ventral line of the 7th abdominal segment ; palpi banded ; 4th and 5th and part or all of the 3rd tarsal segments of the hind legs snow-white.—Ethiopian Region.

3. *Anopheles barbirostris*, Van der Wulp: female with a tuft of scales in the mid-ventral line of the 7th abdominal segment and with scales (in wisps or otherwise) on the venter of all other abdominal segments except the 1st; palpi not banded.—Oriental, extending into Australian Region.

4. *Anopheles umbrosus*, Theobald: female *without any scales* (tuft or otherwise) on the abdomen; palpi not banded.—Oriental (in Malay Peninsula), Ethiopian (in West Africa); presumably restricted in range and not common.

A SYNOPSIS OF THE GENUS *PHLEBOTOMUS*.

By Miss SOPHIA L. M. SUMMERS, M.A., B.Sc.

PHLEBOTOMUS, Rondani.

Phlebotomus, Rondani, Sopro una specie di Insetto Dittero, Memoria prima per servire alla Ditterologia Italiana, 1840, p. 12; Loew, Fauna Austriaca, Dipt., ii. 1864, p. 630. *Cyniphes*, Costa, Ann. d. Acad. degli Aspir. di Napoli, 1843; Schiner, Fauna Austriaca, ii. 1864, p. 631; Grassi, Mem. d. Soc. Ital. Sci. (iii.) xiv. 1907, p. 353; Annandale, Rec. Ind. Mus. (iv.) iii. 1910, p. 40: Newstead, Bull. Ent. Research, ii. 1911, p. 57; Alcock, Entomology for Medical Officers, 1911, p. 115; Brunetti, Fauna of British India, Diptera Nematocera, 1912, p. 199.

Species have been described from all parts of the world, except, at present, Australia.

Phlebotomus, like all the *Psychodidæ* ("moth midges"), has a very hairy body and hairy wings in which the anterior and posterior curvature are very nearly alike, and long filiform antennæ and palpi.

The characteristic features of *Phlebotomus* are as follows:—The body (though thickly covered with long hairs) is distinctly midge-like, the legs also being very long and slender. The eyes and the clypeus are very prominent; the proboscis consists of a fleshy labium (with labella of no very great size), in which the dagger-like epipharynx is most conspicuous; the maxillary palps are long and flexible, and the antennæ are composed of 16 verticillate segments. The wings (fig. 1), which in repose are elevated, are distinguished by the mode of branching of the 2nd longitudinal vein, the bifurcation of which occurs near the middle of the wing (instead of in

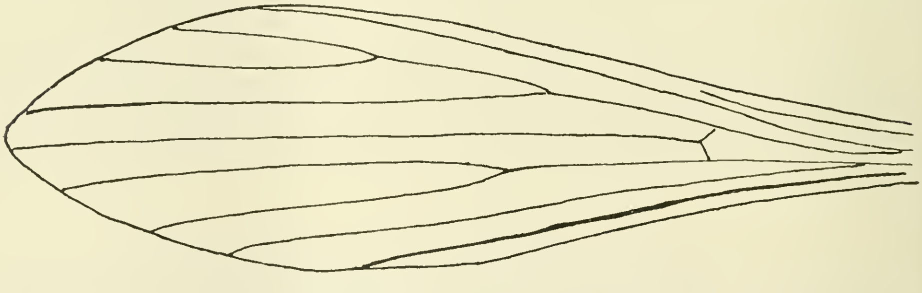

Fig. 1.—Denuded wing of *Phlebotomus minutus*.

the root of the wing), while the bifurcation of the upper branch occurs well in the distal half of the wing. The claspers of the male (fig. 2) are trifid, the dorsal branch consisting of two segments, the distal one of which bears five or four long spinules.

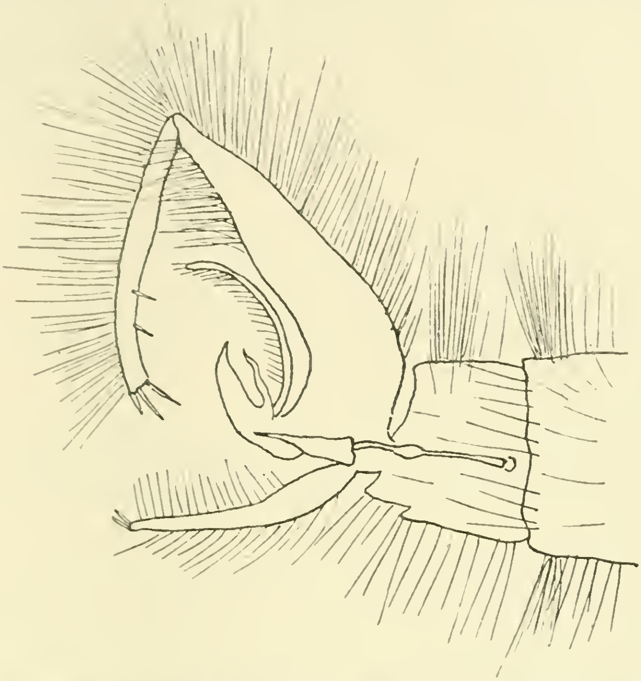

Fig. 2.—Side view (only one side shown) of claspers, etc., of *Phlebotomus papatasii.*

A.—EUROPEAN SPECIES.

Five species have been reported from Europe, viz.:—*P. papatasii*, Scop., *P. mascitti*, Grassi, *P. minutus*, Rond., *P. perniciosus*, Newst., and *P. nigerrimus*, Newst.; but *P. perniciosus* is considered by Dr. Annandale to be a variety of his earlier-described *P. major.*

1. Phlebotomus papatasii, Scop.

Bibio papatasii, Scop. Deliciae fauna et flor. Insubriciae, i. 1786, p. 55; Rondani, Ann. de la Soc. Entom de France, 1843, pp. 265–267. *Cyniphes molesta*, Costa, Ann. d. Acad. degli Aspir di Napoli, 1843; *Haemasson*, Loew, Ent. Zeit. Stettin, v. 1844, p. 115; Schiner, Fauna Austriaca, ii. 1864, p. 631; Grassi, Mem. d. Soc. Ital. d. Sci. (iii.) xiv. 1907, p. 353; Annandale, Rec. Ind. Mus. iv. 2, 1910, p. 51; Alcock, Entomology for Medical Officers, 1911, p. 118; Newstead, Bull. Ent. Research, ii. 1, 1911, p. 73; Brunetti, Fauna of British India, Diptera Nematocera, 1912, p. 211.

Phlebotomus papatasii is a widely distributed species, it having been reported from Europe, Asia, and Africa. It is a large species, about

2·5 mm. in length, and it is covered with yellowish hairs which stand out from the body in little tufts. The thorax has a reddish median stripe and a red spot on each side. The wing in the male is not so broad as in the female. The anterior branch of the second longitudinal vein is longer than the distance between the forks and a little shorter than the distance between the posterior branch and the cross-vein. The hind leg is about 4 mm. long. In the male genitalia the terminal segment of the upper clasper has five long spines, three at the tip and two about the middle of the segment.

2. PHLEBOTOMUS MASCITTI, Grassi.

Grassi, Rend. Acc. Lincei, Roma. xvii. 2° series, 1908, pp. 681–682; Alcock, Entomology for Medical Officers, 1911, p. 118.

This species seems to be very similar to *P. papatasii*, but the five spines of the upper clasper of the male genitalia are very long and falciform.

3. PHLEBOTOMUS MINUTUS, Rondani.

Rondani, Ann. Soc. Ent. France, ii. 1, 1843, p. 263. *Phlebotomus babu*, Annandale Rec. Ind. Mus. iv. 2, 1910, p. 49; Newstead, Bull. Ent. Research, ii. 1, 1911, p. 69; Alcock, Entomology for Medical Officers, 1911, p. 118; Brunetti, Fauna of British India, Diptera Nematocera, 1912, p. 206; Newstead, Bull. Ent. Research, iii. 4, 1912, p. 363.

Phlebotomus minutus is a small fly about 1·5 mm. in length: the female is often 2 mm. long. It is ochreous in colour, and the hairs on the abdomen are recumbent. Second segment of the palps half the length of the third. Wing narrow and bluntly lanceolate, and is divided into similar halves by the third longitudinal vein; upper branch of the second longitudinal vein shorter than the distance between the two forks, which distance is almost equal to the distance between the second fork and the mid cross-vein. Terminal segment of the upper clasper of the male genitalia bears four spines, two apical and two subapical.

4. PHLEBOTOMUS NIGERRIMUS, Newstead.

Newstead, Bull. Ent. Research, ii. 1, 1911, p. 68; Alcock, Entomology for Medical Officers, 1911, p. 119.

"FEMALE.—*Colour.* Head, thorax, and abdomen brownish black; hairs bright ochreous buff, those on the thorax being slightly paler and erect, those on the abdomen recumbent. Basal segment of antennæ dark brown. Palpi pale to dark brown, hairs similar in colour to those on the body. Legs pale ochreous buff, with ochreous-white, *not silvery-white*, refulgence. Wings ochreous buff or dull golden in some lights.

"*Head.* Proboscis long; eyes black, deeply emarginate in front. *Palpi* and *antennæ* very like those of *P. papatasii*. *Legs* very long, femur of hind pair nearly as long as the abdomen; tibia one and one-third times the length of the femur; tarsi longer than the tibiæ by about one-sixth, or nearly as long as the wing; ungues simple. *Wings* with the hind margin strongly arched; sixth longitudinal vein short, terminating near the centre of the hind margin, the length equal to the distance, in a straight line, from its tip to the tip of the third longitudinal vein; the anterior branch of the second longitudinal vein twice the length of the distance between the two forks.

"Length 2·50 mm.

"The black or brownish-black colour of the integument of this insect will serve as a ready means of distinguishing it from any of its allies. It may also be separated from *P. papatasii*, to which it is closely related in its morphological characters, by the shape of the wing and the shorter sixth longitudinal vein." (*Newstead.*)

Loc. Maltese Islands.

Phlebotomus major, var. *perniciosus*, Newstead.

Phlebotomus perniciosus, Newstead, Bull. Ent. Research, ii. 1, 1911, p. 70; Alcock, Entomology for Medical Officers, 1911, p. 118; Brunetti, Fauna of British India, Diptera Nematocera, 1912, p. 211.

"MALE.—*Colour immediately after death.* Eyes black. Thorax with or without dull red-brown spots; when present they are arranged in a triangle, and there is occasionally a similar spot on the vertex of the head. Thorax and coxæ pale, translucent ochreous; abdomen similar, but sometimes pale smoky grey. Hairs pallid. Wings faintly iridescent in strong light, pale drab in subdued light; costal fringe generally very dark or blackish grey, though examples with pale costal fringes are not uncommon. Legs silvery grey, in a strong light presenting a distinct metallic lustre; in certain lights also, those segments which lie in the shadow appear almost black and show up in marked contrast to those which are so placed that their surfaces refract the light. In some lights the under surface of the legs appears distinctly and regularly speckled, a character due evidently to the regular arrangement of the scales.

"*Head* densely hairy, with generally two ill-defined tufts. Clypeus with a large tuft of hairs, some of which are directed forwards, others backwards towards the forehead. *Palpi* with segments 2, 3, and 4 equal in length and collectively a little longer than 5. *Antennæ* with the second segment much longer than the two succeeding ones; the longest hairs on segment 14 almost equal in length to those on the preceding segment. *Thorax* densely hairy, usually with a tuft on the front portion and another on the scutellum. *Abdomen* densely hairy, the longest hairs arising from the apical margin of the segments, but no distinct tufts are found as in *P. papatasii*. The arrangement of the hairs is similar in both sexes, but blackish hairs are often intermixed with the pale ochreous ones on various parts of the body in the darker forms of this insect. *Legs* shorter than those of *P. papatasii*. *Wings* with the posterior border much more strongly arched than the anterior border; the anterior branch of second longitudinal vein nearly as long as the stem between the cross-vein and the proximal fork.

"*External genitalia.* Superior clasper with five very long, stout, curved spines; two apical, one external and two internal placed a little in advance of the outer one; inferior clasper nearly twice the length of the intermediate appendage and clothed to the apex with very long and slender hairs; intermediate appendage somewhat finger-shaped and hairy, proximal portion with a large keel-like extension ventrally, the distal margin of which bears several (5–6) hairs; apex of intromittent organ deeply divided or forked, with occasionally a minute central tooth; exposed portion of the genital filament about half the length of the intromittent organ.

"FEMALE.—With the palpi, antennæ, and legs similar to those of the male. Wings very slightly larger and broader than those of the male.

"Length 1·9–2·2 mm." (*Newstead.*)

It is also stated by Mr. Newstead that the male of this species can be easily distinguished from that of *P. papatasii* by its smaller size, shorter legs, and much smaller genital armature, which is little more than half the width of the abdomen.

Mr. Newstead described this as a distinct species, but Dr. Annandale (Brunetti, 'Fauna of British India, Diptera Nematocera,' 1912, p. 211) considers it merely a variety of his *P. major*.

Loc. Maltese Islands.

B.—ASIATIC SPECIES.

Eleven species have been described from Asia, namely:—*P. papatasii*, Scop., *P. angustipennis*, Meijere, *P. argentipes*, Annandale & Brunetti, *P. babu*, Annandale, *P. minutus*, Rondani, *P. himalayensis*, Annandale, *P. major*, Annandale, and var. *griseus*, Annandale, *P. malabaricus*, Annandale, *P. marginatus*, Annandale, *P. perturbans*, Meijere, and *P. zeylanicus*, Annandale; but of these, in Annandale's later opinion, *P. babu* is a synonym of *P. minutus* and *P. marginatus* a variety of *P. argentipes*, while *P. angustipennis* is thought by Annandale to be a synonym of *P. papatasii*.

1. PHLEBOTOMUS PAPATASII, Scop.

Vide supra.

2. PHLEBOTOMUS ANGUSTIPENNIS, Meijere.

Meijere, Tijdschr. voor Ent. lii. 1909, p. 202; Annandale, Rec. Ind. Mus. iv. 2, 1910, p. 52, and Spolia Zeylanica, vii. 26, 1910, p. 62; Alcock, Entomology for Medical Officers, 1911, p. 120.

Meijere's description of this species is very short. The wings are similar to *P. perturbans*, but the forked cell of the third longitudinal vein has a much shorter stalk. It was described from one spirit-specimen.

Annandale thinks it is synonymous with *P. papatasii*.

3. PHLEBOTOMUS ARGENTIPES, Annandale & Brunetti.

Annandale & Brunetti, Rec. Ind. Mus. ii. 1908, p. 101; Annandale, Rec. Ind. Mus. iv. 2, 1910, p. 44; Alcock, Entomology for Medical Officers, 1911, p. 119; Brunetti, Fauna of British India, Diptera Nematocera, 1912, p. 207.

It is about 2 mm. in length. Head and thorax brownish, abdomen covered with black bristly hairs, and the emargination of the segments often black. Wings narrow, obtusely pointed at the tip, nearly three times as long as broad. Legs very long, hind pair being about three times as long as the thorax and abdomen together; femora much shorter than the tibiæ; metatarsi in all cases at least as long as the remaining tarsal joints combined. In the male genitalia the distal joint of the upper appendages considerably shorter than the proximal one, bearing five stout pointed spines, each nearly as long as the joint—two apical, two near the middle, and one about one-third from the base.

PHLEBOTOMUS ARGENTIPES, var. MARGINATUS, Annandale.

P. marginatus, Annandale, Spolia Zeylanica, vii. 26, 1910, p. 62, and Spolia Zeylanica, vii. 28, 1911, p. 203; Brunetti, Fauna of British India, Diptera Nematocera, 1912, p. 209.

"*Size and Proportions.*—Total length of dried specimen about 2·5 mm. Length of wing 2·5 mm. Hind leg less than two and a half times the length of the thorax and abdomen; its femur about half as long as its tibia, of the same length as the first joint of the tarsus, which is equal in length to three distal joints together.

"*Colour.*—Rather darker than that of *P. zeylanicus*, the costal borders of the wings distinctly darker than their area.

"*Wings.*—Resembling those of *P. zeylanicus* in shape, but longer. The first longitudinal vein only reaching forward for a short distance beyond the anterior fork of the second vein. The anterior branch of the latter about twice as long as the distance between the posterior fork and the point at which the vein joins the third. The fork of the fourth vein distinctly nearer the tip of the vein than the posterior fork of the second." (*Annandale.*)

Described from a single female.

Loc. Peradenyia.

4. PHLEBOTOMUS MINUTUS, Rond.

Vide supra.

Brunetti, in 'Fauna of British India, Diptera Nematocera,' 1912, p. 207, says it occurs in India and Ceylon.

PHLEBOTOMUS BABU, Annandale.

Annandale, Rec. Ind. Mus. iv. 2, 1910, p. 49, and Spolia Zeylanica, vii. 26, 1910, p. 61; Alcock, Entomology for Medical Officers, 1911, p. 119.

Annandale states that this species is synonymous with *P. minutus*, Rond. ('Spolia Zeylanica,' vii. 28, 1911, p. 203).

PHLEBOTOMUS MINUTUS, var. NIGER, Annandale.

P. babu var. *niger*, Annandale, Rec. Ind. Mus. iv. 7, 1911, p. 320.

"Darker than the typical form and, as a rule, larger.

"This form was at first regarded as a distinct species and is marked as probably being so in Mr. F. M. Howlett's collection. I cannot, however, distinguish any constant difference in its venation or genitalia. As yet the variety has only been taken in Bihar. I have not seen it in Calcutta." (*Annandale.*)

5. PHLEBOTOMUS HIMALAYENSIS, Annandale.

Annandale, Rec. Ind. Mus. iv. 2, 1910, p. 50; Alcock, Entomology for Medical Officers, 1911, p. 119; Brunetti, Fauna of British India, Diptera Nematocera, 1912, p. 204.

"*Size.*—Total length 2–2·5 mm. Length of wing 1·5 mm.; greatest breadth of wing 0·4 mm.

"*Colour.*—Yellowish grey with the usual silvery lights; the dorsum of the thorax slightly darker than the sides.

"*Head.*—Rostrum rounded at the tip, stout and elongated. Antennæ normal, with two circles of flat scales and one of flattened hairs on the second joint of the scape. Palpi with five joints, the fifth the longest.

"*Thorax* and *abdomen* as in *P. babu*.

"*Wing* narrow, obtusely pointed, the posterior margin slightly more strongly arched than the anterior; the anterior branch of the second longitudinal vein not much shorter than the second branch, at least four times as long as the distance between the two forks, which is about half as long as that between the posterior fork and the mid cross-vein; the posterior fork and the fork of the fourth longitudinal almost level, the former being slightly nearer the tip of the wing than the latter.

"*Genitalia.*—♀ appendages short and broad, the superior not much longer than the inferior. (♂) Inferior appendage slender, distinctly elbowed, rounded and slightly clubbed at the tip, nearly as long as basal joint of superior appendages, devoid of chitinous spines. Intermediate appendages simple, pointed, curved downwards at the tip. Superior appendages with the distal joint about half as long as the proximal, bearing at the tip three pointed chætæ, each of which is slightly shorter than the joint. Intromittent organ with two elongate, slender valves, which are truncated at the tip." (*Annandale.*)

Loc. Himalayas.

6. Phlebotomus major, Annandale.

Annandale, Rec. Ind. Mus. iv. 2, 1910, p. 46; Alcock, Entomology for Medical Officers, 1911, p. 119; Brunetti, Fauna of British India, Diptera Nematocera, 1912, p. 210.

"*Size and Proportion.*—Total length 3–3·75 mm. Length of wing 2·75 mm.; greatest breadth 1 mm. Hind leg two and a half times as long as the head and body; its femur less than half as long as the tibia and considerably shorter than the first joint of the tarsus, which is longer than the remaining joints together.

"*Colour.*—A uniform golden grey with very strong silvery lights; the disk of the wings with a bluish iridescence; eyes black; legs sometimes darker than abdomen.

"*Head.*—Rostrum somewhat conical, short, truncated in front. Eyes widely separated, emarginate above. Antennæ with the two joints of the scape subspherical: those of the flagellum elongate, slender, densely covered with soft hairs. Palpi with five joints; the basal joint very short, the second, third, and fourth subequal, shorter than the fifth.

"*Thorax* much as in *P. argentipes*.

"*Abdomen* long and slender, clothed with long recumbent hairs and with tufts of longer, upright ones on the dorsal surface.

"*Wings* rather narrower than in *P. argentipes*, pointed, with the posterior border much more strongly arched than the anterior. The anterior branch of the second longitudinal vein much longer than the distance between the second fork and the mid cross-vein: the second fork slightly nearer the base of the wing than that of the fourth longitudinal vein.

"*External genitalia.*—(♂) Superior and inferior appendages similar to those of *P. argentipes*, except that they are more slender and elongate, the basal joint of the superior appendage being nearly twice as long as the last apparent joint of the abdomen; intermediate appendages devoid of a ventral lobe; valves of the intromittent organs slender and blunt: genital filaments well developed.

"This species is closely allied to *P. argentipes*, from which it is easily distinguished by its larger size, uniform golden colour, more elongate tarsi and male genitalia." (*Annandale.*)

Loc. Himalayas.

PHLEBOTOMUS MAJOR, var. GRISEUS, Annandale.

Annandale, Rec. Ind. Mus. iv. 7, 1911, p. 320: Brunetti, Fauna of British India, Diptera Nematocera, 1912, p. 211.

This is a variety of *P. major* in which the colour is darker, usually grey or brown.

Loc. Darjiling.

7. PHLEBOTOMUS MALABARICUS, Annandale.

Annandale, Rec. Ind. Mus. iv. 2, 1910, p. 48: Alcock, Entomology for Medical Officers, 1911, p. 119: Brunetti, Fauna of British India, Diptera Nematocera, 1912, p. 214.

"*Size and Proportions.*—Total length 2·5 mm. Length of wing 2 mm.; greatest breadth 0·5 mm. Hind leg $1\frac{1}{2}$ times as long as the thorax and abdomen: the femur nearly $\frac{3}{4}$ the length of the tibia, $1\frac{2}{3}$ that of the first joint of the tarsus, which is $\frac{3}{4}$ that of the remaining joints together.

"*Colour.*—Thorax, abdomen, and legs (except coxæ and trochanters) brown with a tinge of purple and with the usual silvery lights: wings purplish, strongly iridescent: head, coxæ, and trochanters pale brown: eyes black.

"*Head.*—Rostrum short, rounded in front. Antennæ normal: the second joint of the scape pear-shaped, with several circles of hairs and scales: the first joint of the flagellum much longer than the second. Palpi long, with five joints; the basal joint very short, the second shorter than the third or fourth: third and fourth joints subequal, together shorter than the fifth.

"*Thorax* densely covered with upright curved hairs.

"*Abdomen* covered with recumbent hairs.

"*Wings* pointed; the posterior border not much more strongly arched than the anterior. The anterior branch of the second longitudinal vein very long, about four times as long as the distance between the two forks of the vein, which is much shorter than that between the posterior fork and the mid cross-vein; the fork of the fourth longitudinal almost level with the posterior fork of the second.

"*Genitalia.*—(♀) Much as in *P. perturbans.* (♂) Inferior appendage slender, cylindrical, slightly shorter than the basal joint of the superior appendage, bearing at its tip (in addition to a bunch of fine bristles) seven minute, conical, chitinous spines. Intermediate appendage without a ventral branch; the dorsal branch resembling that of *P. perturbans.* Superior appendage with the distal joint less than half as long as the proximal one, bearing four chætæ, each of which is as long as the joint; one pair of chætæ is situated at the tip of the appendage and the other a short distance nearer the base of the joint. Intromittent organ long and slender: the apices of the valves club-shaped: genital filaments well developed." (*Annandale.*)

Loc. Travancore.

8. PHLEBOTOMUS PERTURBANS, Meijere.

Meijere, Tijdschr. voor Ent. 52. 1909, p. 201; Annandale, Rec. Ind. Mus. iv. 2, 1910, p. 47; Alcock, Entomology for Medical Officers, 1911, p. 120; Brunetti, Fauna of British India, Diptera Nematocera, 1912, p. 205.

A large species about 2·5 mm. in length; colour brown, head, coxæ, and trochanters yellowish. Abdomen covered for the most part with recumbent hairs and a few upright ones both dorsally and ventrally. Breadth of the wing about one-third of its length; length of the second marginal cell about one-fourth the length of the wing. In the male genitalia, the superior appendage is very long and bears four spines, two apical and two at about the middle of the segment.

According to Dr. Annandale it is very similar to *P. malabaricus*, but is distinguished by its dark brownish colour.

Loc. Himalayas.

9. PHLEBOTOMUS ZEYLANICUS, Annandale.

Annandale, Spolia Zeylanica, vii. 26, 1910, p. 60; Brunetti, Fauna of British India, Diptera Nematocera, 1912, p. 215.

"*Size and Proportions.*—Total length of dried specimens about 3 mm. Length of wing 3 mm. Hind leg more than two and a half times as long as the thorax and abdomen; its femur less than three-quarters, but more than half as long as its tibia, slightly longer than the first joint of the tarsus, which is distinctly shorter than the three distal joints together.

"*Colour.*—Head, thorax, and abdomen brown, paler in the female than in the male: coxæ yellowish; femora, tibiæ, and tarsi silver-grey; wings pale brownish grey, uniform in colour.

"*Wings.*—Lanceolate, not very broad, with the two borders not unlike in curvature. The first longitudinal vein extends far forwards, overlapping the anterior branch of the second vein by more than three-quarters of its length. This branch is nearly five times as long as the distance between the two forks, which is much shorter than the distance between the posterior fork and the point at which the vein joins the third vein. The fork of the fourth vein is almost on a level with the posterior fork of the second. The course of the sixth vein, which bends down almost at an angle at the tip, is sinuous.

"*Male Genitalia.*—The distal joint of the superior appendage is slightly shorter, and much more slender than the proximal joint; its outlines are somewhat sinuous, and it bears five long, stout, curved, sharp chætæ, which are arranged as follows:—A pair at the tip of the appendage, a pair on the outer margin at about half the length of the joint, and a single chæta on the inner margin nearer the base. The chætæ are equal or subequal. The intermediate appendage (morphologically the lower branch of the superior one) is slender, pointed, and turned upwards at the tip. It bears a minute, pointed, naked lobe on its ventral surface, and a similar one on its external lateral surface. The inferior appendage is much longer than the proximal joint of the upper one: it is slender as viewed from the side, and of almost uniform width; the tip is narrowly obliquely truncate, and bears a brush of very long and slender hairs; the rest of the appendage is sparsely covered with rather shorter hairs, but there are no spines.

"The genitalia of this species closely resemble those of *P. argentipes*, from which it is distinguished among other characters by the venation of the wing. The venation closely resembles that of *P. malabaricus* (from Travancore) and *P. himalayensis*, but the insect is paler than the former and darker than the latter species. Its genitalia are also very different from those of either." (*Annandale.*)

Loc. Peradeniya.

C.—AFRICAN SPECIES.

There are five species described from Africa, but two of these (namely, *P. papatasii*, Scop., and *P. minutus*, Rond.) are common to Europe and Asia.

1. PHLEBOTOMUS PAPATASII, Scop.

Vide supra (under European species).

2. PHLEBOTOMUS MINUTUS, Rondani.

Vide supra (under European species).

PHLEBOTOMUS MINUTUS, var. AFRICANUS, Newstead.

Newstead, Bull. Ent. Research, iii. 4, 1912, p. 363.

"Length (average) 2·25 mm.; largest wing, ♀ 2·28 mm., ♂ 1·95 mm.; smallest wing, ♀ 1·78 mm., ♂ 1·33 mm.

"This is clearly a well-defined race of *Phlebotomus minutus*, Rond., differing in the relatively longer second and fourth segments of the palpi in both sexes, and in its more pointed wings. It is also, as a rule, larger, but there is a marked range of variation in size, so that this character alone cannot be taken as a guide to its identification. It is, moreover, generally darker than typical *P. minutus*, but in all other respects, including the structural details of the male genital armature, it is inseparable from the latter. It may be noteworthy to add that Annandale has described a variety of *P. minutus*, Rond., from India, under the name *niger*, and says that it is 'darker than the typical form and as a rule larger,' though he failed to 'distinguish any constant difference in its venation or genitalia.' No reference is made to the structural characters of the palpi, neither has he noted any difference in the form of the wing. I am forced to the conclusion, therefore, that the African race is not synonymous with the var. *niger* of Annandale.

"MALE.—Colour generally much more dusky than that of typical examples of *P. minutus*, Rond. Upper portion of thorax and abdomen pale smoky-brown; thoracic hairs erect, dusky greyish, with brownish tips; abdominal hairs on first and second segments long and erect, forming two small tufts dorsally; remaining hairs shorter, recumbent and generally uniformly dark brown, being usually much darker than the integument. Wing-fringe generally smoky-grey with an admixture of darker hairs.

"*Proboscis* very slightly shorter than the head, inclusive of the clypeus. *Palpi* with the segments 2 to 4, inclusive, each successively longer; second segment one-third to one-fourth longer than the third; fifth very variable in length, but usually three to three and a half times the length of the second. *Antennæ* with the third and fourth segments relatively shorter than those in *P. minutus.*

"*Wings* lanceolate and markedly pointed; tip of first longitudinal vein either opposite to, or considerably in advance of, the anterior fork of the second longitudinal vein, so that this character is given to great variation.

"FEMALE.—*Palpi* with all the segments usually slightly shorter than those of the male." (*Newstead.*)

Loc. West and East Africa.

3. PHLEBOTOMUS DUBOSCQUI, Neveu-Lemaire.

Neveu-Lemaire, Paris Bull. Soc. Zool. 31, 1906, p. 65; Picard, Bull. Soc. Ent. France, 1909, p. 169; Austen, African Blood-sucking Flies, 1909, p. 20; Alcock, Entomology for Medical Officers, 1911, p. 119; Newstead, Bull. Ent. Research, iii. 4, 1912, p. 366.

Colour pale yellow—about the same size as *P. minutus.* Wing narrow, about three times as long as broad; 2nd marginal cell less than a third the length of the wing. In the female the antenna is composed of thirteen segments, and the last segment of the palps is shorter than the second last segment. Male genitalia similar to *P. papatasii.*

4. PHLEBOTOMUS ANTENNATUS, Newstead.

Newstead, Bull. Ent. Research, iii. 4, 1912, p. 365.

"Length 2·35 mm.; wing 1·4 mm.

"This species may be distinguished at once by the short stout form of the antennal segments, of which the third to the thirteenth, inclusive, are much more bead-like than those of any other species which has hitherto been described. Furthermore, it may be distinguished from *P. minutus*, var. *africanus*, by its shorter and stouter legs.

FEMALE.—Colour and arrangement of the hairs on the head, thorax, and abdomen somewhat doubtful, owing to the rubbed condition of the specimen, but their arrangement appears to be the same as in *P. minutus.*

"*Antennæ* with all the segments unusually short and stout; the third scarcely one-third longer than the fourth; the thirteenth segment only very slightly longer than the succeeding one; the paired and geniculated spines are present on the third to the fifteenth segments, inclusive; the hirsute glands, seen so clearly in other species, not traceable, and if present they are apparently quite rudimentary. The long hairs on all of the segments decidedly scattered and not so much confined to the proximal portions as is usually the case.

"*Palpi*: second to fourth segments, inclusive, like those of *P. minutus*, including also the compound groups of spines upon the proximal portion of the third segment; length of terminal segment doubtful, as one of them is wanting and the other is rendered partly obscure by the superimposed legs, though it is apparently shorter than the corresponding segment in *P. minutus. Legs* relatively short and stout. *Wings* similar to *P. minutus*, but all the veins are relatively stouter." (*Newstead.*)

Loc. Gold Coast.

5. PHLEBOTOMUS SQUAMIPLEURIS, Newstead.

Newstead, Bull. Ent. Research. iii. 4, 1912, p. 366.

"Length 2·65 mm.; wing 1·84 mm.

"FEMALE.—Pleuræ *clothed with large flat mosquito-like scales*. This character alone readily distinguishes this insect from all other known members of the genus.

"*Colour*: integument dull brown (possibly a very variable character in museum specimens). Head and thorax densely hairy, the hairs dusky grey: abdominal hairs recumbent and similar in colour to those on the thorax: they are, however, somewhat sparse. Wings beautifully hyaline and, in certain lights, the hairs on the fourth longitudinal vein golden iridescent: fringe greyish, posteriorly very long and dense.

"*Head*, inclusive of clypeus, equal in length to the proboscis, the latter, especially the labium, somewhat slender. *Palpi* very long and somewhat slender: third segment one-sixth longer than the second: fourth segment scarcely longer than the third: fifth scarcely twice the length of the fourth: segments 2–4 inclusive without any marked incrassation; some of the few remaining scales which clothe the palpi are of great size and broadly lanceolate, but for the most part these organs are denuded in the microscopical preparation. *Antennæ*, with the exception of the first two segments, wanting, but those which remain are somewhat thickly clothed with minute, short hairs. Legs long, but fairly stout; anterior and mid-trochanters spinose, the spines short and arranged in groups. Wings decidedly narrow, much more so than in the female *P. papatasii*." (*Newstead.*)

Loc. Anglo-Egyptian Sudan.

D.—AMERICAN SPECIES.

Three species have been described from America:—*Phlebotomus vexator*, Coquillett, *Phlebotomus cruciatus*, Coquillett, and *Phlebotomus rostrans*, Summers.

1. PHLEBOTOMUS VEXATOR, Coquillett.

Coquillett, Ent. News, 18, 1907, p. 102; Alcock, Entomology for Medical Officers, 1911, p. 120.

Length 1·5 mm. Yellow species with brown mesonotum and hairs; legs are also brown, but covered with a white tomentum; first submarginal cell is slightly over twice as long as its petiole. The terminal segment of the upper clasper of the male genitalia has five spines—two apical, two subapical, and one at the middle of the segment.

Loc. Plummers Island, Maryland.

2. PHLEBOTOMUS CRUCIATUS, Coquillett.

Coquillett, Ent. News, 18, 1907, p. 102; Alcock, Entomology for Medical Officers, 1911, p. 120.

It is similar to *Ph. vexator*, but can be distinguished by its yellow hairs and by the first submarginal cell, which is about three times as long as its petiole. Male unknown.

Loc. Cacao, Treca Aguas, Guatemala.

3. PHLEBOTOMUS ROSTRANS, Summers.

Summers, Bull. Ent. Research, iii. 2, 1912, p. 209.

Described from specimens mounted in Canada balsam, so that the colour is uncertain. The hairs that remain on the abdomen are long and recumbent. *Head* of the female is long, half the length of the rest of the body; clypeus more than ordinarily prominent, its length being about equal to that of the rest of the head. In the hind pair of *legs* the tibia is as long as the combined head and body. *Wings*: in the male the greatest length of the wing is three and a half times its greatest breadth, bluntly pointed, and the hind border not much more strongly arched than the front border. The length of the second marginal cell is contained $2\frac{2}{3}$ times, and that of the third marginal cell $1\frac{5}{6}$ times, in the length of the wing. In the female the wings are decidedly broader, but the relations of the veins are about the same. Halteres large and very long. The terminal segment of the upper clasper of the male genitalia has four stout spines, one terminal, one ante-terminal, one rather beyond the middle of the supero-lateral border, and one a short distance behind the middle of the ventral border.

This species can be easily distinguished by the great length of the head. The wing-venation is similar to *P. malabaricus*, Annandale.

Loc. Rio Javary, South America.

Note.—In the Yellow Fever Bureau 'Bulletin' for January 1913 there is a short reference to a paper by A. Lutz and A. Neiva, published in Mem. Inst. Oswaldo Cruz, 1912, iv. 1, pp. 84–95, in which three new species of *Phlebotomus* from Brazil are described. The writer has not had an opportunity of seeing this paper.

II.—DEPARTMENT OF PROTOZOOLOGY.

A Further Note on a Case of Dermal Leishmaniasis from S. America, with the Results of Inoculation Experiments.

(Successful Inoculation of a Cat.)

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In a former number of this Journal (Vol. I. Pt. 3) I described a case of dermal leishmaniasis from S. America. The case was one of an Englishman who had contracted the disease in Peru or Bolivia, where he had been travelling. He had two sores—one a large one over the right eye, and another smaller one at the outer corner of the same eye. The present note deals with the subsequent history of this case after its treatment was commenced, the result of the culture of the leishmania obtained from it, and some inoculation experiments on animals.

Treatment.—A reference to my former paper will show that the case was treated in two ways. In the first place, killed cultures of the leishmania were injected subcutaneously. This treatment did not appear to be checking the disease, as the smaller sore showed signs of spreading towards the eye. Accordingly a second mode of treatment was adopted in the form of applications of an ointment made of equal parts of methylene-blue, lanoline, and vaseline, as recommended by Cardamatis and Melissidis. The ointment was applied on a piece of lint, the size of the sore, both morning and evening. This caused very little pain, at any rate nothing that the patient was unwilling to endure. The effect of the ointment was to cause a great increase in the exudation, which was of liquid consistency and of a dark blue colour. The ointment was applied to the small sore at the outer corner of the eye for two weeks, and, as it showed signs of healing, the applications were stopped. In another fortnight healing was complete. To the larger sore over the eye treatment with the ointment was continued for two and a half months, and, as it was thought that possibly the ointment had killed the leishmania but was preventing healing, the treatment was stopped and a simple ointment applied. In two weeks' time the sore had completely healed. Since then the patient has been seen from time to time. He was last seen ten months after the sores had healed, and there was no sign of any recurrence. The glandular enlargement, which was such a marked feature of the case, had steadily diminished and had practically disappeared, though one of the nodules under the skin of the right eyebrow could still be detected, though it was much reduced in size. The resultant scarring was very slight compared with the extent of the lesions, and this would have been still less had not an attempt been made to excise the larger sore before the patient came under treatment and the condition had been diagnosed.

It is to be hoped that this treatment with the methylene-blue ointment will be given a good trial, for in the present case it has had a very pleasing result.

Inoculation Experiments with the Cultures of the Parasite.

In the earlier paper I described how a culture of the leishmania was obtained on N.N.N. medium. The growth in this medium was very luxuriant, so that it was easy to maintain the culture by subinoculating tubes. This was successfully carried out for seventeen generations in N.N.N. medium, so that there is every probability that, as with other strains of leishmania, it would be possible to carry on the culture indefinitely. Compared with a culture of *Leishmania tropica*, obtained from the Oriental Sore of Aleppo, the South American leishmania grew much more readily, though morphologically no constant differences could be made out between the two types of parasite.

With the cultures some inoculation experiments were made :—

1. With the first culture obtained (Nicolle's culture virus) a human being was inoculated on the skin of the forearm in two places—one by intracutaneous injection with a hypodermic needle, and the other by scarification. A year has now elapsed since the inoculation and no infection has taken place.
2. A young rabbit was inoculated intravenously on two occasions with rich cultures of the leishmania, but no infection took place.
3. A young rabbit was inoculated intravenously on one occasion with rich culture, but did not become infected.
4. Attempts were also made to infect mice by intraperitoneal injection of rich cultures, but without success.

Experiments with the Virus from the Sore.

I previously described the successful inoculation of a dog and baboon directly from the sores on the man :—

1. The baboon was inoculated in the skin on each side of the nose and on the tips of the ears by means of a hypodermic syringe. After an incubation-period of four months, sores containing leishmania had appeared on the ears and nose. The sore on the nose was in the form of a papule which persisted for about two months and finally disappeared without ulceration. The lesions on the ears were in the form of small papules which ultimately reached the size of a pea, and, owing probably to scratching by the monkey, finally ulcerated. They persisted for six months, and then gradually disappeared, finally healing at the end of the seventh month. Shortly after this the monkey died from some unknown cause, but no sign of leishmania could be found in its organs.

On August 22, 1912, a black dog was inoculated on each ear in several places with material obtained by scraping one of the sores on the baboon. After twenty-three days three small papules had appeared on the dog's left ear. In these leishmania were discovered. The incubation-period was thus much shorter than in the case of the baboon inoculated directly from the man. Later nodules appeared on the other ear. These increased slightly in size, but had disappeared in five months.

2. In my former paper I recorded the successful inoculation of a dog (i) directly from the man. A sore developed on each ear after an incubation of two months. One sore was excised for inoculation purposes, the other was left. Though seven months have elapsed since its appearance, it has not yet disappeared, but is now represented by a shallow ulcer. The excised sore was emulsified with saline solution and injected into each ear of another dog (ii). After an incubation-period of one month dog (ii) had developed a small nodule on one ear, and two weeks later a similar nodule on the other ear. One month later one of these nodules was excised and emulsified with saline for inoculating other animals (*a*, *b*, *c*). The remaining one was left alone. It persisted for three months and then absorbed. From the emulsion of the sore the following animals (*a*, *b*, *c*) were inoculated :—

a. Small puppy intravenously. The animal, previously healthy, began to lose weight and had a temperature up to 102·1° F. during the next month. It was in such poor condition that a general infection was suspected. During this time, however, liver puncture (twice) and bone marrow puncture (once) gave only a negative result as regards leishmania. Two months after inoculation, the dog appeared to be improving in health, so was killed. Smears from its organs were negative, as were also six tubes of N. N. N. medium inoculated from its spleen.

b. Two mice inoculated intraperitoneally did not become infected.

c. A cat inoculated in each ear developed small pinhead nodules after two months at the sites of inoculation. These have increased slightly in size, and now, five months after inoculation, are still present, while leishmania have been discovered in them first by the culture method on N. N. N. medium, and later by microscopical examination of smears from the sore.

Experiments on the Transmission of *Trypanosoma lewisi* by means of Fleas.

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In the last number of the 'Journal of the London School of Tropical Medicine,' in a paper on the behaviour of *Leishmania tropica* and allied flagellates in bugs and fleas, I mentioned that I had been able to confirm Nöller's results on the transmission of *T. lewisi* by means of the dog-flea, *Ctenocephalus canis*. These experiments were conducted by feeding the fleas, which were fixed on wires, as Nöller has recommended, first upon myself in order to determine by examination of the fæces passed by the fleas during the feed that they were uninfected; then feeding them upon an infected rat, and subsequently upon myself until the characteristic small infective trypanosomes of Swellengrebel and Strickland had appeared in the fæces, which were received upon a cover-glass held in a suitable position while the flea was feeding. The fæces, which are liquid, are at once made into a thin film with the edge of another cover-glass. The dried film is then stained with a Romanowsky stain, and any infection of the flea is at once detected. When the flea was infected with the small trypanosomes it was allowed to feed upon a clean rat, and the fæces passed during the feed were collected as before upon a cover-glass, but instead of allowing them to dry they were taken up at once with a fine pipette in some suitable liquid, as, for instance, the liquid in a tube of N. N. N. medium. The diluted fæces were then transferred to the mouth of another clean rat, care being taken not to touch the mouth with the pipette for fear of injuring the skin. This experiment was repeated three times with the dog-flea, and in each instance it was the rat which received the fæces per os that became infected in either six or seven days, while the rat upon which the fleas fed did not become infected. These experiments are in agreement with those of Nöller, who concluded that rats become infected with *T. lewisi* not by the bite of the flea but by licking up the infective fæces deposited by the fleas upon their fur.

This experiment I have since repeated with the human flea, *Pulex irritans*, in which I have previously shown that *T. lewisi* will undergo a complete development up to the appearance of the small infective trypanosomes in its fæces. In the case of *Pulex irritans* the fleas were also fixed upon wire after Nöller's method. I have further made attempts to transmit *T. lewisi* by means of *Pulex irritans* when free. On three occasions, fleas which were found to be infected by examination of fæces were liberated from the wire and placed upon an uninfected rat. In every instance the flea disappeared, but whether it had died or had been eaten by the rat I could not determine. The fact that the rat in no case became infected is rather an argument against the latter view. It appeared then that *Pulex irritans* does not like to live upon rats. I have since made many attempts to induce *Pulex irritans* to live upon white rats, but in every instance that a flea is placed on the rat it exhibits the greatest uneasiness and continues to jump on and off the rat, giving one the impression that it does not feel at home. Its behaviour is in marked contrast to that of the true rat fleas *Ceratophyllus fasciatus* or *Xenopsylla chæopis*, both of which fleas immediately hide themselves in the fur and appear quite satisfied with their lot. This being the case, it cannot frequently happen in nature that *Pulex irritans* transmits *T. lewisi*, and it is remarkable that, in spite of this fact, *T. lewisi* will complete its development in the flea, as shown by the fact that the forms in the fæces are infective.

I have also conducted experiments with *Xenopsylla chæopis* obtained from Bombay rats. Four of these fleas were caught on a rat infected with *T. lewisi*, and, together with one *Ceratophyllus fasciatus*, were transferred to an uninfected white rat on December 9, 1912. The next day (Dec. 10) one of the *Xenopsylla* and the *Ceratophyllus* were removed and fixed upon wires, the three remaining *Xenopsylla* being allowed to remain upon the rat. Both the *Xenopsylla* and *Ceratophyllus* were wired, and were found to have fæces infected with infective forms of *T. lewisi* when fed upon myself. Unfortunately both these fleas died before any crucial experiment could be undertaken to test the infectivity of their bites. The subsequent history of the three remaining *Xenopsylla* is of interest in showing that this flea is a true host of the rat trypanosome. The following passages with the fleas were made :—

1. December 9, 1912. One *Ceratophyllus fasciatus* and four *Xenopsylla chæopis* (3 females and 1 male) were caught on wild rats infected with *T. lewisi*. They were placed on a white rat (1). On December 10, one female *Xenopsylla* and the *Ceratophyllus* were removed for other experiments, as related above, while three *Xenopsylla* remained on the rat. On

January 3, 1913, the rat (I) had a small infection of reproducing forms of *T. lewisi.* The blood of the rat (I) had been examined every alternate day. The period for infection to take place was 25 days. One *Xenopsylla* had disappeared.

2. On January 11, two *Xenopsylla* (1 male and 1 female) were removed from rat (I) and placed upon rat (II). On January 22 this rat had reproductive forms of *T. lewisi* in its blood. This was probably the third day of infection, judging from the number of trypanosomes present in the blood. Period for infection about 9 days.

3. On February 3 the two fleas were removed and placed upon a young rat (III). On February 14 the rat was infected. Period for infection 11 days. One flea (the male) had disappeared.

4. On February 20 the single remaining female *Xenopsylla* was placed upon an adult rat (IV). Two days later this flea had disappeared. The rat did not become infected.

These experiments clearly demonstrate that the common rat-flea of India, *Xenopsylla cheopis*, is a true host of *Trypanosoma lewisi.* The irregularity in the length of the periods elapsing before infection takes place suggests that the infection is due to some chance, such as the licking up by the rat of infected moist flea fæces. If infection takes place only when the rat licks up the moist fæces of the flea (I have shown that the dry fæces is not infective), a considerable element of chance is involved. In those cases where I have transferred the fæces directly it is passed by the flea (*Ctenocephalus* and *Pulex*) to the mouth of the rat, the incubation-period has been very constant and varying only from six to seven days. If the infection was produced by the bite of the flea, then it might be expected that the incubation-period would be similarly constant. The results obtained with the *Xenopsylla* are in favour of the fæcal mode of infection as obtains in the case of the dog and human fleas.

Professor Minchin, who, in conjunction with Dr. Thompson, has conducted experiments with *T. lewisi* and *Ceratophyllus fasciatus*, has suggested that infection takes place by the regurgitation of the small trypanosomes which have wandered forwards from the rectum to the mid-gut. It has been argued that this wandering forwards of the small trypanosomes of Swellengrebel and Strickland must have some meaning, and that it is a preparation for a future regurgitation into the rat. It must be an exceedingly difficult matter to prove that such a regurgitation never takes place, but that it does not do so regularly is borne out by Nöller's experiments and my own, whereby fleas (*Ctenocephalus* and *Pulex*) were made to feed upon rats with every precaution to prevent the rat taking up any flea fæces. In none of these cases has the bite infected, though the fæces have, so that, as far as we know at present, no satisfactory infection experiment has been made wherein the fæcal method of infection has been excluded.

As regards the passage forwards of the small trypanosomes from the fleas' rectum to the mid-gut, there is another—and, to my mind, more probable—explanation. As I have explained in an earlier paper (Jl. Lond. Sch. Trop. Med. vol. ii. no. i.), during the feeding act of the flea its gut is in a state of most remarkable activity. This can very readily be observed by allowing a flea to feed upon one's wrist and viewing it with a hand-lens by means of transmitted light from an electric lamp. It will be seen that the gut is performing the most extraordinary writhing motions, and that violent waves of peristaltic contraction are passing along it, first in one direction and then in another. The result of this action is that when a flea feeds, for instance, on a rat in whose blood *T. lewisi* occurs, some of these trypanosomes escape alive in the first fæces passed by the flea. The contractions have been of sufficient strength to drive some of the live trypanosomes from the mid-gut into the hindmost part of the rectum. It would be a remarkable thing, then, if these movements of the gut did not also have the effect of driving some of the small trypanosomes from the rectum to the mid-gut, seeing that the whole intestinal content is subjected to such a churning action as that described. The passage forwards of the small trypanosomes can thus be looked upon as in the nature of an accident, and not necessarily associated with any definite mode of infection.

Another point which appears to strongly support the fæcal method of infection is the fact that the final development of the infective forms of *T. lewisi* takes place in the rectum rather than in some anterior portion of the alimentary tract. In the case of *Glossinæ* and leeches, which are known to transmit trypanosomes by their bite, the chief or final development is in some anterior part of the gut or its appendages (proboscis, proboscis-sheath, or salivary glands). The site of this development is evidently associated with the method of infection through the bite-wound. Surely, then, the fact that in the flea the main development takes place in the rectum affords great presumptive evidence that infection takes place by way of the hind gut, as indeed, experiments so far conducted seem to show.

During the past three months I have constantly observed the habits of the fleas, *Xenopsylla,* while upon the white rats. Their favourite haunt is the posterior part of the rat's back, just above the root of the tail. Very soon after being placed upon the rat the fleas find their way to this area and commence to deposit their fæces, which appears as black spots upon the white fur. The rat is constantly turning round its head and licking this region of its body, so much so that it seems remarkable that the fleas escape being devoured by the rat. The rat must repeatedly lick up dry fæces of the flea and occasionally the moist fæces just deposited. Another habit of the flea is again in favour of this ingestion of moist fæces. I have often noted that when a flea is quite undisturbed while feeding, it will become distended with blood to an enormous extent. It is then in a condition of unstable equilibrium, for the slightest disturbance will cause it to eject at once a comparative large drop of fæces. A flea feeding thus quietly upon a rat, while the latter is perhaps sleeping, is suddenly disturbed by the awakening animal, who immediately turns to lick its fur and so has every chance of taking up the freshly passed fæces which the irritated flea has deposited.

The fact that *Trypanosoma lewisi* is able to complete its development in so many different Invertebrate hosts suggests that other trypanosomes may follow it in this respect. It is known that *Trypanosoma gambiense* can be transmitted by different species of *Glossina*, and it may be that this and other trypanosomes will be found capable of completing their development not only in flies belonging to different species but even to different genera, as is the case with *T. lewisi* in fleas.

Another important point is the outcome of these results. It cannot any longer be held that a difference in Invertebrate host necessarily means a difference in species of trypanosome. The fact that in one locality a trypanosome is transmitted by a certain fly, while in another locality a similar trypanosome is transmitted by a fly belonging to a different genus, is not a proof that the trypanosomes are different. If, however, it could be shown that the trypanosome in one locality was incapable of development in the fly of the other locality, then it could be fairly safely assumed that the trypanosomes were really distinct. Such a test could with profit be applied to the two human trypanosomes *T. gambiense* and *T. rhodesiense*. The former, we know, is capable of development in both *Glossina morsitans* and *Glossina palpalis*, while the latter has only been shown to develop in *Glossina morsitans*. If it could be shown that *T. rhodesiense* is incapable of development in *Glossina palpalis*, a further strong argument in favour of the distinction of the two species of human trypanosome would be obtained.

A FURTHER NOTE ON *TRYPANOSOMA RHODESIENSE* FROM THREE CASES OF HUMAN TRYPANOSOMIASIS.

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AND

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IN the preceding number of this Journal (Vol. II. Part 1) an account was given of some preliminary observations made by us on three strains of trypanosomes, derived from three cases of human trypanosomiasis from Rhodesia. These cases were the subject of a paper by Dr. Daniels in that number of the Journal. The strains were labelled G, E, T, in correspondence with the human cases from which they were obtained *.

Observations were made on the trypanosomes as they occurred in man and in white rats. A careful search through blood films taken from the human cases failed to reveal any "posterior-nuclear" forms, although in one of the patients the trypanosomes occurred in the blood in rather large numbers.

These forms were found, however, in the blood of the first rats inoculated by us from the human cases; it may therefore be assumed that the strains were truly those of *T. rhodesiense*. Stephens and Fantham, in describing this trypanosome, claimed that the appearance of "posterior-nuclear" forms in the blood of rats inoculated from man was a distinctive character of this particular trypanosome. Since then, Wenyon has shown that "posterior-nuclear" forms occurred in the blood of rats inoculated with *T. pecaudi* from the Soudan, and Blacklock that they occurred also in *T. brucei* (Uganda) and *T. equiperdum*.

Our observations were continued with the object of determining in the subinoculated white rats the constancy of the appearance of the posterior-nuclear forms, their percentage number and the extent of variation of this number, and their relation to the virulence of the strain.

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* Cases G and E are dealt with in this number. Case E, *cf.* abstract of Werner's paper Case G, *cf.* Newham.

To ascertain these points, blood-films were made from the rats every three days or more often. The films were made in the ordinary way, dried in air, and stained subsequently by the Eosin-Azur Romanowsky modification. Starting from the day on which it was found that the trypanosomes in the rat were numerous enough to render counting fairly easy, counts of one thousand trypanosomes were made every three days and the number of "posterior-nuclear" forms occurring in each thousand noted. To facilitate counting a ruled eye-piece scale was made use of, and as the film was moved along under the objective only those trypanosomes coming into view between certain lines on the scale were counted.

Dividing forms with two trophonuclei, of which one was displaced posteriorly, were not seldom seen. They were not counted as "posterior-nuclear" forms. Only those forms were considered as "posterior-nuclear" which showed a *decided* displacement of the single trophonucleus towards the non-flagellar end of the trypanosome. In most instances the nucleus lay either very close to the kineto-nucleus or was separated from the latter only by the vacuole.

We found that though this nuclear displacement occurred most often in broad trypanosomes with short free flagellum, yet others of quite normal dimensions showed this morphological peculiarity, as did also a small percentage of the very narrowest forms with long free flagellum. More or less complete translation of the relative positions of the kineto- and trophonucleus within the trypanosome was observed once in each strain.

The general outward shape of the trypanosomes in the blood-film from the rat varied greatly in some different films in the same strain and in a few instances in the same film. Observation proved this to be due to differences in the thickness of the films, the rapidity with which they were spread, and the time taken in drying them. No conclusions could be based on these appearances.

The results obtained were controlled by the examination of three separate strains from G, one of which (G B) had been started previously by Dr. Newham in rats and then maintained in rats and guinea-pigs for 4 months. The strains were found to resemble each other in all essential particulars.

The Constancy of the "posterior-nuclear" Form in the Blood of White Rats.

This form occurred in all the strains. In all of them also it was at times absent—within the set limits of observation.

In the fifth passage of strain E, during the twelve days the rat lived, no posterior-nuclear forms were found. The parasites were numerous enough to make counting easy on the 6th day of inoculation, and apparently continued to increase in numbers up to the animal's death.

In the T strain the posterior-nuclear forms were absent once—in the fourth and eighth passages respectively. This strain showed consistently the highest percentage of these forms.

In the G A, G B strains the posterior-nuclear forms were most often found to be absent. In every passage investigated it was absent on one or more occasions. These strains showed constantly the smallest percentage of posterior-nuclear forms. In neither strain was the first passage investigated, in G A the 3rd passage also was not investigated, while G B had passed through rats and guinea-pigs for 4 months before coming under examination by us.

The occasional absence of the posterior-nuclear form in all the strains, and its more frequent absence in the G strain, could not be correlated with any other observed phenomenon.

The Percentage of posterior-nuclear Forms and its Variation in the Blood of White Rats.

In all the strains, and in some particular passages, the percentage (based on a count of one thousand trypanosomes) was found to vary greatly. A general survey of the results obtained gives the distinct impression, so far as the scope of this investigation provides warrant for such, that with any given strain of *T. rhodesiense* posterior-nuclear forms are not only more numerous proportionally (and probably absolutely as well, though no total counts were done) in the first passage in the rat, but also tend to become scantier with each subsequent passage.

One may observe also a tendency, sometimes marked, for the posterior-nuclear forms to increase in proportional number *pari passu* with the length of life of the rat. Exceptions, however, can easily be noted.

The investigation has not been exhaustive enough to determine the mean limits of variation, and we consider that no definite conclusion can be based on these results.

In the G A strain the posterior-nuclear forms varied from 0 to 0·9 per cent., in the G B strain from 0 to 4·3 per cent.

In G C, however, in the first passage (not examined in G A and G B) these forms varied from 0 to 8·1 per cent.

In the E strain they varied from 0 to 7·2 per cent., and in the T strain from 0 to 40 per cent.

The Virulence of the Strain, and its Relation to the Presence and Percentage Number of posterior-nuclear Forms.

All the strains show an almost equal virulence, while, as has been noted above, they differ widely, as between each other and within each self, in the constancy and percentage number of posterior-nuclear forms.

Counting from the day of inoculation, the rats with the G A strain lived from 12–30 days, the average for 8 passages observed being 18 days. In the G B strain the rats lived from 15 to 39 days, the average for 6 passages observed being 23·3 days. In G C the first rat lived for 42 days, the number of posterior-nuclear forms being higher than was observed in any other passage from G. It is perhaps noteworthy that this occurred in the first subinoculation direct from the human case.

In the E strain the rats lived from 11 to 38 days, the average being 22·8 days for the 8 passages observed.

In the T strain the rats lived from 16 to 34 days, the average for the 8 passages being 22·75 days.

It is plain that, within the limits of this enquiry, there is no correlation between the number of posterior-nuclear forms and the virulence of the strain for white rats.

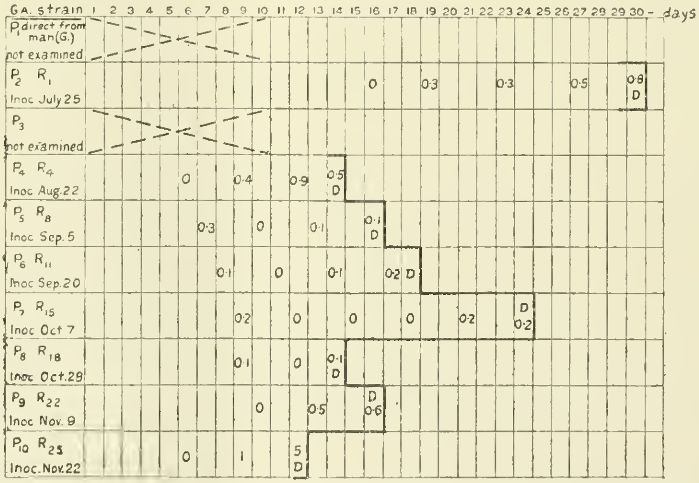
Wenyon, in his paper describing the presence of posterior-nuclear forms in the blood of a white rat inoculated with *T. pecaudi* from the Soudan, noted that in the dividing forms one daughter trypanosome was frequently a posterior-nuclear form, while the other was of the normal type. This phenomenon was often seen in the films examined in this enquiry.

It occurred to us that there might be some direct relation between the proportional number of posterior-nuclear forms and the rate of multiplication of the trypanosomes. No detailed investigation was made on this point. While counting for posterior-nuclear forms a general note was made of the number of dividing forms. Later, separate counts of 1000 trypanosomes each were made on a series of films from the several strains, in which the posterior-nuclear counts had already been done—the number of dividing forms being this time recorded instead.

No constant relation at all was found between percentage of dividing forms and percentage of posterior-nuclear forms.

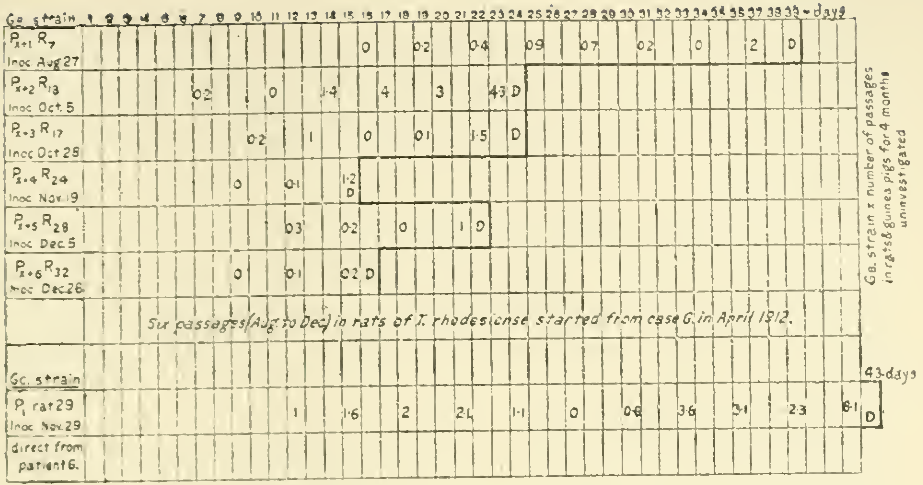
This result is entirely inconclusive, for in order to determine this point daily counts at least should be undertaken, and perhaps even more than one count per day: and controlled by total counts. However this may be, though no total counts were done, in all the passages in every strain, judging from examination of fresh blood preparations and stained films, the trypanosomes increased in number, with perhaps some pauses in multiplication, up to the death of the rat, and, as has already been noted above, there did appear some tendency for the percentage number of posterior-nuclear forms to increase *pari passu* with the length of life of the rat.

The appended tables give further details of the investigation. The figures in the tables give the percentage of posterior-nuclear forms, based on a count of 1000 trypanosomes, on certain given days after inoculation.

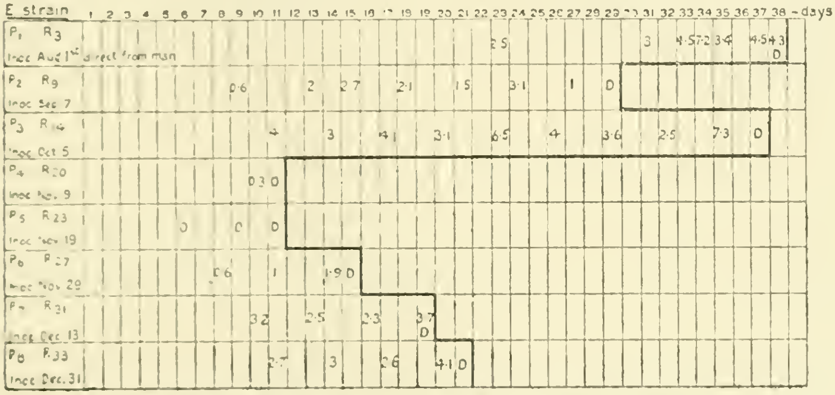


G.A. strain	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	- days
P_1 direct from man (G.) not examined																															
P_2 R_1 Inoc July 25																0			0·3				0·3				0·5			0·8 D	
P_3 not examined																															
P_4 R_4 Inoc Aug. 22						0			0·4			0·9		0·5 D																	
P_5 R_8 Inoc Sep. 5							0·3			0			0·1			0·1 D															
P_6 R_{11} Inoc Sep. 20								0·1			0			0·1			0·2	D													
P_7 R_{15} Inoc Oct 7									0·2			0			0			0			0·2			D 0·2							
P_8 R_{18} Inoc Oct. 28									0·1			0		0·1 D																	
P_9 R_{22} Inoc Nov. 9										0			0·5			D 0·6															
P_{10} R_{25} Inoc. Nov. 22						0			1			5 D																			

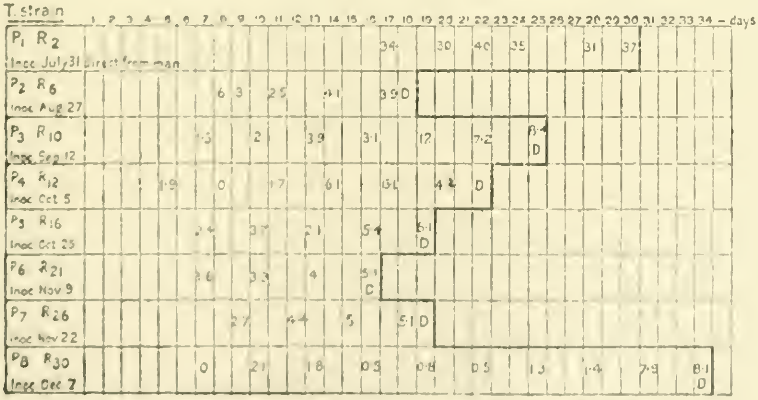
The first 10 passages in rats of Trypanosoma rhodesiense started from case G. in June 1912.



The first passage in the rat of *Trypanosoma rhodesiense* started from case G. Nov. 1912.



The first 8 passages in rats of *Trypanosoma rhodesiense* from case E.



The first 8 passages in rats of *Trypanosoma rhodesiense* from case T.

In conclusion, we would point out that it is significant that for *T. rhodesiense*, even in subinoculations in the rat, it is easy to miss the posterior-nuclear forms, unless somewhat lengthy examination be made of films prepared at frequent intervals from the rat. This fact is of some value in considering the reliance to be placed for purposes of diagnosis on the presence or absence of posterior-nuclear forms in the blood of the subinoculated rat.

It has been noted above that these forms are now known to occur in *T. pecaudi* (Soudan) and *T. brucei* (Uganda).

Recently Stephens and Blacklock have expressed the belief that *T. brucei* of Uganda is not the same species as *T. brucei* of Zululand, the original source of the type species of Plimmer and Bradford. They base their belief on the fact that *T. brucei* (Zululand) is a constant monomorphic trypanosome, while *T. brucei* (Uganda) is a markedly polymorphic one—in this resembling *T. pecaudi* and *T. rhodesiense*.

If this be so, it will probably be found, as some have maintained, that the Uganda *T. brucei* is the same as *T. pecaudi* of the Soudan. The trypanosome of Uganda hitherto known as *T. brucei* would therefore have to be called *T. pecaudi*. Further, there seems to be an accumulating amount of evidence in favour of regarding *T. rhodesiense* as identical with the *T. brucei* of Uganda. If this turns out to be correct, then the specific name *rhodesiense* will have to give place to the name *pecaudi*.

H. da Rocha-Lima on Epizootic lymphangitis and Histoplasmosis.

[Beitrag zur Kenntnis der Blastomykosen. Centralbl. für Bakt. i. Abt., Originale Bd. lxvii. 1912, Heft 4.]

In this paper the author discusses the nature of the parasites discovered in Epizootic lymphangitis of horses and Histoplasmosis of man.

In the case of *Cryptococcus farciminosus*, the parasite of the disease in horses, the author draws attention to the following facts :—

1. The appearance of the living organism with its double contoured membrane, its variation in size and shape, the fact that its ends may be round or pointed, and the character of the contents of its thick envelope, all remind one rather of a yeast-cell than of any other structure.
2. Reproduction by budding described by various authors is also in favour of the yeast view.
3. In the positive Gram staining reaction it again agrees with the yeasts.
4. Like the yeasts the organisms will withstand without destruction the application of concentrated acetic, sulphuric, hydrochloric, and nitric acids.
5. From the lesions various authors have been able to cultivate Blastomycetes, while Sansfelice claims to have observed the parasite under the microscope develop into hyphæ.
6. Bidré and Nègre have tested the reaction of complement fixation, using the serum of infected animals and the *Cryptococcus* or yeasts as antigen. A positive result was obtained in these cases, though the controls with

Bacteria (*B. coli*) and Protozoa (*T. vespertilionis* and *Leishmania infantum*) were always negative.

The author then records his own observations upon material obtained from cases of the disease in horses. He finds that the lesions produced do not resemble those of Kala Azar nor Oriental Sore, and that the resemblance to these diseases is only a superficial one, in that the parasites are taken up by large cells. A similar condition can be produced by the inoculation of yeasts into the tissues of animals. In reaction to Romanowsky stain the organisms behave like yeasts and not like leishmania. The large red mass appearing within parasites stained in this way corresponds to the similar red mass in yeast-cells, and represents rather the shrunken protoplasm or metachromatic material than a true nucleus like the large nucleus of a leishmania. Sometimes within the *Cryptococcus* a deep red staining granule appears besides the larger red mass. This has been compared to the small nucleus of the leishmania, but it is not of constant appearance, though it may represent the true nucleus of the parasite. It thus appears that there is every reason to regard the *Cryptococcus farciminosus* as of the nature of a yeast.

The author then turns his attention to a consideration of *Histoplasma capsulatum*, the parasite described by Darling from the organs of three Chinamen who had died of some obscure disease in Panama. The disease was characterised by wasting, irregular remittent fever, large spleen, and anæmia with leucopenia, while pathologically there was an invasion of the endothelial cells of the small lymph- and blood-vessels by an enormous number of parasites, which produced necrosis and cirrhosis of the liver, enlargement of the spleen, pseudogranulomata in the lung, and in the intestine ulceration with involvement and necrosis of the corresponding lymphatic glands.

The author finds that the *Histoplasma capsulatum* agrees in every way with the *Cryptococcus farciminosus* considered above. The marked variation in size, the character of the parasite with its thick envelope and contents, its Gram positive staining reaction, the presence of forms evidently reproducing by budding, and the type of lesion produced by its action, all speak in favour of this view, while the presence of the parasite within phagocytes and a certain resemblance to leishmania when stained by Giemsa stain are points quite insufficient to establish the protozoal nature of the organism. Both the *Cryptococcus* and the *Histoplasma* are to be regarded as of the nature of yeasts.

C. M. W.

Cutaneous Phagedœna caused (?) by *Entamœba tetragena*.

A. Carini contributes to the 'Bulletin de la Société de Pathologie Exotique' (10th April and 11th Dec., 1912) two interesting papers calling attention to this little-known * complication of amœbic liver-abscess. Carini writes from S. Paulo, Brazil, where he had observed two cases (which he describes), and had learnt from other physicians and surgeons in that place that the affection was not extremely rare.

It was only found as a complication following on multiple abscesses of the liver due to amœbæ in very cachectic subjects on whom operation had been performed late in the disease ; the operation-wound and surrounding skin having been constantly bathed in abundant pus containing large numbers of amœbæ. The lesion started by a redness around the margins of the operation-wound, followed in a few days by the appearance in this red area of greyish necrotic spots, which spread until there were extensive areas of gangrenous sloughs separated from healthy skin by a zone of intense inflammation. The condition was little amenable to treatment. Large areas of the skin and subcutaneous tissue, down to the fascia covering the muscles, over the abdomen and thorax were destroyed. The pus from this area, the sloughs, and inflamed tissue were found full of amœbæ. In the sections of inflamed tissue the amœbæ were seen surrounded by a dense leucocytic infiltration. Carini examined films of the pus, wet-fixed by Schaudinn's solution and stained with iron-hæmatoxylin. The amœbæ, he states, presented the characters of *Entamœba tetragena*.

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* Dr. C. W. Daniels has observed cases such as those described by Carini.—*Private Communication.*

As a rule, the cases ended in death ; one of Carini's cases, however, recovered. In the literature he had been able to find only one record of such an affection—that was a case described in 1908 by Ménétriés and Touraine (Sol. Med. des Hôpitaux).

It is interesting to note that apparently before the amœbæ can produce this extensive necrosis of skin and subcutaneous tissue, it is necessary that the tissues be not only constantly bathed in pus, and thus presumably damaged to some extent, but that their normal power of resistance be lowered as a consequence of the marked cachexia of the patient.

Wenyon, in the preceding number of this Journal, described his recent work on the experimental production of amœbic (*E. tetragena*) dysentery in cats ; and from those researches and his former observations on the entamœba of mice (*E. muris*) came to the conclusion that *E. tetragena* could not, in all probability, invade the healthy mucous membrane of the gut, until the epithelial cells had been damaged. The damage in this case being probably the result of a toxin produced by the amœba.

Incidentally, also, the possible occurrence of the grave complication recorded by Carini indicates that in that type of case operation for abscess of the liver had better be performed by the method of trocar and canula ; for the open operation, as in Carini's cases, must entail a constant bathing of a wound in weakened tissues, by pus which may contain large numbers of pathogenic amœbæ. H. M. H.

III.—DEPARTMENT OF HELMINTHOLOGY.

In the absence of

R. T. LEIPER, D.Sc., M.B., &c.

Dr. R. T. Leiper, the Helminthologist to the School and the first Wandsworth Scholar, is engaged on an expedition in Nigeria to investigate the human helminthes. The first-fruits of the expedition are highly satisfactory, as he has proved that the carrier of *Filaria loa* is a Chrysops and that two species, *Chrysops dimidiata* and *Chrysops silacea*, serve this purpose. In both cases the embryos (*Filaria diurna*) developed in the salivary glands of these biting flies.

The date of his return is not yet decided, but we hope to have a contribution from him for the next issue. C. W. D.

HELMINTHOLOGICAL NOTES.

Some recent Researches on Human Filariasis and Elephantiasis.

F. Ouzilleau, in an interesting paper [" Les Filaires humaines de la région du M'bomou (Afrique équatoriale française). Pathogénie de l'éléphantiasis de cette région : Rôle de *Filaria volvulus*." Bulletin de la Société de Pathologie Exotique. Tome 6. No. 1. Jan. 8th, 1913], records observations, which, if confirmed in other parts of Africa, form an important addition to our knowledge of the etiology of Elephantiasis and the part played by *Onchocerca* (*Filaria*) *volvulus* in human pathology.

We give here an abstract of his paper :—

On the Mbomou River (one of the two affluents at the head of the Ubangwe) the proportion of people suffering from elephantiasis varied from 0·3 to 3 per cent., according to district. The lesions observed were : elephantiasis proper of the penis, scrotum, labium majus and minus, and the legs : lymph scrotum and adeno-lymphocele.

Brumpt, who had given a classical description of *F. volvulus*, did not believe that the parasite had any pathogenic action. The author, however, after a year's practice in the Mbomou region, during which time he had examined a large number of natives, had noted that the parasite was the cause of many ailments, among which elephantiasis was the most striking. In his investigation of the etiology of elephantiasis, the researches of Manson had led the author to study first the local filariases. He soon confirmed

Bernard's observations that the human filariases in that region were *Filaria perstans*, *Filaria loa*, and *Filaria volvulus*, and that *Filaria bancrofti* was not represented. Ouzilleau had never found this worm in the 1500 blood-examinations made during the day and the 400 made at night.

The endemic index for *F. perstans* was 64 per cent. in natives over 10 years old. It was found associated with *F. loa* in 30 per cent. He had never found it in pure lymph-gland juice, nor had he ever found the adult form. The embryos were found in 45 per cent. of cases infected with *Filaria volvulus*, whether suffering from elephantiasis or not.

For *Filaria loa* the endemic index was 16 per cent. It was found in 10 per cent. of cases infected with *F. volvulus*. It seemed thus that the presence of *F. volvulus* was unfavourable to infection with *perstans* and *loa*, for the embryos of these last two species were found in a greater proportion of cases free from *volvulus* infection.

Microfilaria loa was found rarely in lymph-gland juice.

Ouzilleau here quotes Bernard, who punctured the inguinal glands of 5 subjects on whom he operated for elephantiasis and claimed that he had found "filariæ with very distinct sheaths (m. f. diurna ?) : their presence was perhaps due simply to blood being mixed with the lymph."

The author, practising methodical inguinal-gland puncture, had obtained quite different results.

The adult *Filaria loa* was often found. Bernard had found several specimens encysted in the tunica vaginalis in operations for hydrocœle, and had suggested the possibility that *F. loa* might be the cause of hydrocœle in that region. Ouzilleau had always found his specimens of *loa* alive. They were found often in operations on the genitalia (elephantiasis, hydrocœle, hernia). In one case of hernia, six were found—there was no effusion into the sac, the parasites were lying between dartos and fascia near the testis or higher up on the cord. No relation could be established between *F. loa* and *F. perstans* and the lesions of elephantiasis in that part of the country. It was noteworthy that only 45 per cent. of cases of elephantiasis were infected with *F. perstans* and 10 per cent. with *F. loa*, while the average in non-elephantoid cases was 64 per cent. for *perstans* and 16 per cent. for *loa*. Again, the geographical distribution of those Filariæ and that of elephantiasis did not coincide. The former were common throughout Equatorial Africa ; elephantiasis, on the other hand, was somewhat rare and had a very limited area of distribution. It was necessary, then, to look elsewhere for the causal agent in elephantiasis.

Filaria volvulus alone remained for study in its relation to this disease. From the time of his first arrival, the author's attention had been directed to this point by Dr. Bernard, who had himself reported that it was worthy of note that the geographical distribution of elephantiasis corresponded with that of *F. volvulus* and that most of the cases of that disease were affected at the same time with the tumours of *F. volvulus* ('Rapport de Dr. Bernard sur l'installation des effectifs dans la région de Zémio,' Oct. 1911), and again pointed out this fact in his annual report 1911.

Ouzilleau eventually found that 43·3 per cent. of cases of elephantiasis also had *volvulus* tumours. In the large number of blood-examinations he had made during his researches on *F. perstans* and *F. loa* he had never found any other filaria than those two. One day, after performing resection of an inguinal tumour in a case of elephantiasis, films were made from the lymph-gland tumour. They revealed the presence of microfilariæ identical with those found in *volvulus* cysts: they must have reached the glands along the lymphatics.

From this time on, 79 operations were performed for elephantiasis, adenolymphocœle, or lymph scrotum. A careful examination was made in each case, and as a result the author could make the definite statement that all subjects suffering from elephantiasis or from like tumours were infected with *Filaria volvulus*. Of the 79 patients, 35 had both the adult *F. volvulus* in cysts on the surface of the body, and its embryos in enlarged inguinal lymph-glands. In 44 the embryos were found in the inguinal glands, while the tumours were not noticed. It was found afterwards that these tumours could be easily missed.

The author, in consequence, thought the rule justified that in every case of elephantiasis the embryos of *Filaria volvulus* could be found in the inguinal lymph-glands.

(Later, one case was observed in which the tumours were present, but no embryos were found in either blood or gland-juice.)

The adult F. volvulus.

The adults were found in cysts or tumours, generally superficial, situated under the skin. Adhesions were often present between tumour and underlying tissues, and this was especially the case in the neighbourhood of bone where the tumours were often incorporated with the periosteum. In 80 per cent. of cases the tumours were on the sides of the thorax; they might be so small as to be found only by careful palpation of the whole body; they could often be found in places where bony prominences or articular surfaces were in close contact with the skin. The cysts were often found in the course of operations—in the inner half of the groin, on the symphysis pubis, along the spermatic cords, all places in which they could easily be mistaken for lymphatic glands. They were also found in the abdominal wall. The author thinks that in the 55 per cent. of cases in which only the embryos were found in the lymphatic glands the cysts containing the adults were too small to be seen, or were hidden in thickenings over bones or situated deep in the thoracic or abdominal cavity. However, the absence of such affections as chylocœle, chyluria, etc., which were found in cases of Filariasis due to *F. bancrofti*, indicate that *F. volvulus* and its embryos did not select the deep lymphatics, in which places they could not fail to produce lesions like those they caused at the surface (elephantiasis, adenitis, etc.). The conclusion must be drawn that the *volvulus* cysts situated near joints do not differ markedly from those tumours known as "juxta-articular nodes." Brumpt had observed many cases of these nodosities in Uganda and some on the Ouelle (which forms with the Mbomou the head-waters of the Ubangwe). In every case of this sort seen by the author he had punctured the tumour and found the embryos, and sometimes fragments of the adult, of *F. volvulus*.

The embryos of Filaria volvulus.

Besides the fluid contained in the cysts, the place of selection for the embryos was the lymphatic network in the inguino-crural region. The glands containing the embryos were often very much enlarged (hen's egg). Puncture of these glands (with the technique of gland-puncture in trypanosomiasis) revealed their presence generally at the first go—invariably at the third. The embryos were seen swimming about in the midst of leucocytes, They swarmed in adeno-lymphocœle lesions. The author had never observed enlargement of the axillary and epi-trochlear glands. In a very few cases he found the embryos in enlarged glands at the acromio-clavicular joint and in the cervical glands ; and most times he had found them in subjects who showed no elephantoid lesions but rather some general ailment. Out of 200 blood-examinations (day or night) the embryos were found in the blood on one occasion only. This patient had a *volvulus* tumour on the thorax. The blood examined showed very numerous embryos of *F. volvulus* ; they were also found in the fluid from the tumour and in the inguinal gland-juice.

Ouzilleau states that :—

1. Every individual infected with *F. volvulus* has not got elephantiasis nor must necessarily develop it.
2. But such an individual always suffers from an inguino-crural adenopathy (the glands containing *F. volvulus*), which most often remains a simple adenitis, but which sometimes develops into adeno-lymphocœle.
3. This adenopathy may persist for a long time without causing any apparent impairment of health. Often, however, it is complicated with general ailments and local lesions more or less grave in character.

The embryos of *F. volvulus* were always present alive, and without sheaths, in the lymphatic system of infected persons and patients with elephantoid lesions.

The lymph stasis was caused by living embryos ; and the inflammation and fibrosis of the lymphatics were the result of irritation set up by the embryos, either mechanically or by the secretion of some toxin.

Though microbic agents might play some part in certain attacks of fever occurring in elephantiasis, there was nevertheless no need to always pray in aid their intervention. The Filariæ were themselves quite capable of provoking both the local inflammatory changes and the general febrile state.

Ouzilleau is convinced that : " la *Filaria volvulus* doit être incriminée en tant qu'agent pathogène de l'éléphantiasis local (M'bomou) à l'égal de la *Filaria bancrofti* dans l'éléphantiasis des Arabes."

The author promises a forthcoming paper recording further observations. Those interested in the subject are advised to consult the original paper.

Meanwhile, it should be noted that Ouzilleau's researches apply, just now, only to the M'bomou region. A search (admittedly not exhaustive) among the literature on elephantiasis and onchocerciasis in West Africa has not revealed to the writer any observations other than Ouzilleau's and Bernard's (quoted by Ouzilleau) on the coincidence or otherwise in area of distribution of *Onchocerca volvulus* and elephantiasis or elephantoid lesions. Both worm and disease, however, are recorded from a rather wide extent of the West African regions : though it seems that insufficient data are at hand to enable one to indicate with any approach to exactness even some of their respective areas of distribution (and the same may be said of *F. bancrofti* in that country). Ouzilleau's observations can hardly fail to stimulate research on this matter, as well as on the biology of *Onchocerca volvulus*.

It may be noted that Labadie-Lagrave described the parent *volvulus* worms as lying in lymphatics which were surrounded by fibrous tissue. Fülleborn, though unable to confirm this definitely, thought it at least not unlikely.

H. M. H.

NOTES ON BILHARZIOSIS.

Its antiquity.—Professor A. R. Ferguson (Glasgow Med. Jl., Jan. 1913, Vol. 29, No. 1) states that since the earliest times Bilharziosis has existed in the Nile Valley: for (1) Calculus of the urinary bladder is found so often in mummies of every dynasty as to suggest the operation of the same local cause as now exists, viz. bilharziosis; (2) Hæmaturia must have been very common among the ancient Egyptians, for they knew of and practised many kinds of remedies for this affection; (3) Ruffer has recently demonstrated calcified bilharzial ova in microscopical preparations from the kidney of a mummy of one of the later dynasties.

Its spread in recent times.—Since the Philippine war bilharziosis was now found in many parts of the United States of America. In the Lane lectures, delivered in San Francisco in 1905, Manson gave warning that this might happen. Ferguson himself felt certain that soldiers returning from the South African war must have introduced the disease into Australia.

W. Horace Nelson records that this has actually happened. In the 'Australasian Med. Gazette,' Nov. 9, 1912, he describes three cases of bilharziosis: two of the patients had never been outside of Australia—in their case the disease must have been contracted in that country. One of the patients, a woman, must have acquired the infection, in some way or other, from an ex-soldier from the Transvaal, who was storekeeper at the timber-mill where the woman lived. Rain-water was used for drinking. The water for the mill was pumped from a place over a hill a mile away. There were no bathing facilities. The ex-soldier had to micturate almost every half-hour, and his fingers, soiled with urine, may have infected the bread he handled.

The second indigenous case was a West Australian male, 21 years old, who lived 12 miles away. From the contour of the country it was impossible that drainage from the mill could reach there. However, more than a year before the time of onset of his symptoms, he had been at the mill for a period of two weeks. He was a platelayer, and may have acquired his infection from the flushings of a lavatory-car used by an infected person.

The assumption appears to be, following the theory of Looss, that infection is direct from man to man, and no intermediate host is necessary in the life-history of the *Schistosomum*. Indeed, Ferguson and Davy (*loc. cit.*) believe that they have evidence supporting the view that the miracidia enter man through the nasal mucous membrane and perhaps also at the genitalia.

The miracidia live from 30 to 40 hours only in water, and they are killed in 3 minutes by 1 in 2000 solution of HCl—so that, so far as direct infection is concerned, the stomach at least must be ruled out. It is equally well known that the inhabitants—savage, or even civilised—of endemic areas connect the acquisition of the disease with the use of water in or from certain local rivers, ponds, etc.: a popular belief which has recently received confirmation, for Katsurada has shown that cats can be infected with *Schistosomum japonicum* by immersing their legs in the water of such suspected streams. Leiper failed to infect a monkey by injecting under its skin water containing large numbers of active miracidia of *S. hæmatobium*.

Bionomics of the Bilharzial Miracidium.—G. Bouet and E. Roubaud record some interesting experiments with the miracidia of *Schistosomum hæmatobium* in Dahomey and Haute-Casamance (Bull. Soc. Path. Exot. Dec. Vol. 5, No. 10).

All the schistosome ova found by them, in urine or fæces, were terminal-spined.

Such ova kept in urine for 12 to 20 hours at 23°–25° C. set free mobile miracidia when placed in water. The miracidia did not survive in ova kept in pure urine for more than 20 hours.

Clots containing ova were divided into two lots : one lot was placed in pure water and the embryos emerged in less than one hour ; the other lot was placed on slightly moistened sand. The embryos did not emerge until these ova were placed in pure water 12, 24, and 30 hours later.

Ova were placed on a little bed of sand the periphery of which could absorb some water from a small surrounding moat. 30 hours later living miracidia were found in the water surrounding the bed of sand.

The authors point out the biological importance of the property possessed by the embryos of keeping alive for more than 24 hours in ova placed in damp sand. In the act of micturition, eggs might be deposited on damp soil near collections of water ; and the miracidia set free from them, by the first shower of rain occurring within 30 hours, could reach the water either by their own efforts or by being just washed there.

The authors confirm Conor's findings that the embryos do not survive drying of the ova. Eggs, naked or covered with clot, dried for a few minutes did not set free their embryos. Miracidia exposed to drying for only a few minutes do not revive when placed in water.

Conor, in Tunisia, showed that the Bilharzia miracidia remained alive and active at 45° C. and only died quickly after 50° C. The optimum temperature was between 39° and 40° C. for the Tunisia miracidia. Bouet and Roubaud, experimenting with the embryos in Casamance, found that after 41° C. they were barely alive ; after 45° C. they died in less than 5 minutes. At 41° C. many died after 15 minutes. A few lived on for 1¼ hours, contracted up like a ball and very sluggish. On putting them back into a lower temperature a few revived.

The Casamance miracidia appeared to have an optimum temperature of 30° to 35° C.

Conor claimed that the distribution of Bilharziosis in Tunisia was determined by the presence of springs of warm water (42° to 45° C. at Gabès). There would thus appear to be in Tunisia a geographical race of *Schistosoma* characterised by the high degree of resistance to heat possessed by its embryos.

The authors found that a trace of HCl or soap in the water quickly killed the embryos.

"*Fixation*" *of the Miracidia in water to which serum is added.*—A few drops of dog's serum were added to water in a watch-glass containing active embryos. Evaporation of this albuminous fluid produced at the edges a thin semi-solid border. All the miracidia, one after the other, butting against this slightly viscous border, stop, thrust the anterior end several times into the viscous mass as if searching for a favourable spot, and then become quiescent. A minute later the peripheral cilia and the flame-cells cease to move. This phenomenon is seen if the water is mixed with a few drops of urine containing blood, and appears to be a biological fixation such as is undergone by the embryo at the start of its development in its host. Nothing like it is seen in pure water, even during continuous evaporation. The phenomenon has nothing in common with the entanglement of the embryos in sticky matter. Embryos enclosed in mucus move about trying to get free. Even when progression is prevented, the cilia continue to move, and when these at length cease, the cilia of the flame-cells can be seen moving for a still longer period.

The authors, however, failed to observe any analogous behaviour of embryos placed on pieces of skin and mucous membrane of monkeys, rats, and fowls, or directly on the bodies of small rodents. They failed to infect rats and mice soaked for many hours in water teeming with embryos. They confess themselves unable to form any idea of the normal mode of infection of human beings.

H. M. H.

GAPES IN MAN: a Notice of its Discovery by Dr. A. King in St. Lucia.

LEIPER, writing from Calabar, records in the 'Lancet' (Jan. 18, 1913) that just before leaving England he had received from Dr. A. King, St. Lucia, specimens of a male and female *Syngamus*. The worms were *in copula*. Being away from typical material for comparison, Leiper does not name the species. The parasites were obtained from a woman who had suffered from chronic cough and had coughed up, she stated—the worms with some blood. Leiper thinks it very probable that this *Syngamus* is a purely accidental infection of man with a parasite common in one of the domesticated animals. *Syngamus trachealis* is a very common parasite of poultry and game-birds. He, however, knew of no undoubted instance of the infection of a mammal with a normally avian parasitic nematode. Unless Dr. King had been misled by a neurotic patient, the find was most interesting and suggestive.

H. M. H.

IV.—CLINICAL MEDICINE.

A Case of Raynaud's Disease with Extensive Gangrene.

By H. B. NEWHAM, M.R.C.S., L.R.C.P., D.P.H.

In view of the fact that extensive gangrene as a sequel to Raynaud's disease is of rare occurrence and is usually only seen in children, a report of a case showing this condition in an adult may be considered worthy of being placed on record.

The patient (J. J.), aged 25, a native of Grenada, West Indies, was admitted to the Seaman's Hospital, Albert Docks, on December 14th, 1910. He shipped as a fireman on board a steamer bound for London—this being the first occasion on which he had left his native land.

He gave the following history:—He was in perfect health till 15 days previous to admission, and stated that he had had no previous illness. He then suddenly noticed, on coming on deck, that his fingers and toes became very cold, he lost all sensation in them, and power of movement was lost. Warming them before a fire did not restore warmth or sensation.

On the next occasion when he passed urine he noticed that the same was quite black. This passage of dark urine from time to time continued for some 24 hours, and then the urine cleared and remained so onwards.

He had to give up all work and lay in his bunk till the ship reached London.

The patient had no rigor, no lumbar pains, no vomiting or diarrhœa. He suffered from acute pains down the legs and arms and on touching or pressing the skin just above the limit of the area of loss of sensation in the hands and feet.

He did not notice any discoloration of the hands or feet during the time he was confined to his bunk, but ascribes this to the fact that the cabin in which he lay was very dark.

He stated that he suffered from great thirst for 10 days before admission to hospital, but had no soreness of gums or tongue.

Condition on admission.

A well-nourished man. Tongue covered with slight moist fur. Lips covered with dry sordes. Teeth good and gums healthy. Eyes showed nothing abnormal.

Thorax.—Pigeon-chested. Heart and lungs normal.

Abdomen.—Liver and spleen not palpable. Nothing abnormal detected. Bowels constipated. No rash on the trunk.

Legs.—On both limbs, from the toes upwards to 1½ inches above the bend of the ankle, the skin was very dark, almost black and somewhat glossy; the feet were swollen and cold, but no definite œdema. The patient felt deep pressure and could move foot and toes at ankle, but there was a total loss of sensation over the skin of the affected areas, except over a ring some half inch in depth, where the skin was practically quite black, and situated between the normal skin of the leg and the dark brown cold skin of the foot. This black ring was markedly hyperæsthetic.

Above the affected area the legs appeared normal, except for slight pain on deep pressure—otherwise sensation was normal. Knee-jerks were present and normal. On the dorsal aspect of the left great toe there was a small abrasion.

Hands.—On both hands the three outer fingers were black, cold, and with a narrow ring of hyperæsthesia at the line of the metacarpo-phalangeal joints.

The index-fingers on both hands showed patchy, cold, black areas devoid of all sensation.

The terminal phalanges of the three outer fingers on both hands were glossy, black, and shrivelled, with complete loss of sensation. The patient could move his fingers, but could only feel on deep pressure.

The loss of sensation and gangrenous areas appeared confined to the skin, whilst the deeper tissues (except in the case of the terminal phalanges of the three outer fingers of each hand) appeared to be unaffected.

The thumbs on both hands appeared healthy. The extremities of the ears were unaffected, but there was a small gangrenous patch the size of a 3*d.* piece on the tip of the nose.

The urine was of normal colour—sp. gr. 1010—no albumen, sugar, or blood. The temperature on admission was 98°, but rose a few hours later to 101°·2 F., and remained at about this height, with slight intermissions, but never reaching normal, for some five days, when it fell to normal and with occasional rises of a degree or so remained normal onwards.

A differential and total count of the blood gave the following figures :—

Red Cells	3,900,000 per c.mm.
White Cells	25,000 ,,
Hæmoglobin	70 per cent.
Lymphocytes	9 per cent.
Large Mononuclears . . .	5 ,,
Polymorphonuclears . . .	86 ,,

No parasites were found, and examination of the stools revealed nothing abnormal.

By January 2nd, some three weeks after admission, a well-marked line of demarcation was present on both legs two inches above the ankle on either side, and both feet appeared entirely dead as regards both deep and superficial tissues.

On February 11th it was decided to amputate the right foot and the fingers of the right hand.

The patient was placed under an anæsthetic and the right leg was amputated about halfway between the knee and ankle.

At the same time, the fingers of the right hand were removed at the metacarpo-phalangeal joints, and the heads of the metacarpal bones were excised. The thumb was preserved.

On February 22nd a second operation was performed of a similar nature, and the left leg and left fingers were removed.

The patient stood both operations well and the further history of the case was uneventful, the man ultimately leaving the hospital on August 15th in good general health.

LIVER-ABSCESS.

By GEORGE M. GREY, M.D., F.R.C.S.,

WEST AFRICAN MEDICAL STAFF.

LIVER-ABSCESS is one of the most serious maladies which Europeans in the Tropics are liable to, the mortality in cases surgically treated being about 20 per cent. The varieties of this disease, as met with on the West Coast of Africa, may be classified into three divisions: acute, subacute, and chronic. The latter variety has, in my experience, occurred mostly in natives, whilst the acute and subacute types were found in Europeans. During my last tour of service in Southern Nigeria my attention was directed by a case which occurred in Lagos Hospital to another type of liver-abscess, fulminating in character, and to which Herrick* has applied the term, "Very severe toxic." The details of this particular case were as follow:—

E. W., a European locomotive driver on the Lagos Railway, was admitted to Lagos Hospital on March 29th, 1912. On the evening of March 19th, while at Offa, he felt feverish and out of sorts, and took a dose of ten grains of quinine. He had a restless night, but in the morning he went on duty and took his train to Jebba. On his arrival there he was seen by the Medical Officer, his temperature was found to be 103° F., and he was put on the sick list. Next morning he complained of pain in the epigastrium, and quinine had no effect on the temperature, which remained continuously high. During the next two days the temperature remained up, and the pain was so severe as to require hypodermic injections of morphine. On the 25th the patient was sent down to Ibadan.

For the details as to the patient's condition at Ibadan I am indebted to Dr. Finlay, medical officer in charge there. The temperature was 102°·6, the tongue was furred, and complaint was made of continuous pain in the epigastric region, the upper right rectus muscle was rigid. No enlargement of the liver was made out, and the spleen was not palpable. The pulse numbered 92 per minute, was small and regular, and the heart-sounds were normal. A slight cough was present and a few snoring rales could be heard over both lungs generally. In the anterior axillary line, at the level of the sixth rib, rough crepitations could be heard at the end of respiration and the expiratory murmur was wheezy. The pain complained of in the epigastric region was increased on deep inspiration. The urine was deep amber in colour, acid in reaction, contained a trace of albumen but no blood. A blood-examination revealed no parasites, and a differential count resulted as follows:—

Polymorphs	74	per cent.
Lymphocytes	17	,,
Large Mononuclears	7	,,
Eosinophiles	2	,,

The patient was put on 5 grains of Pulv. Ipecac. Co., every four hours, and hot fomentations were applied to the site of the pain. On the 26th pain and fever were continuous, but no fresh symptoms were observed. On the 27th the liver-dulness seemed to be slightly increased. A saline mixture was given every half hour and the bowels acted well after six doses, the motions were watery and pale yellow in colour. The patient's condition remained practically unchanged for the following two days, the temperature being remittent in character, rising as high as 103° F., and the pulse averaging 100 per minute. On the 29th of March Dr. Finlay sent him down to Lagos Hospital with a diagnosis of suspected liver-abscess.

The patient was admitted under the charge of Dr. A. Hutton, who was acting Resident Medical Officer. On admission to hospital his temperature was 104°·6 and his pulse was 116 per minute. A slight bulging in the right hepatic region was noted and the area of hepatic dulness was increased. The patient looked very ill, and on the morning of the 30th Dr. Hood, Deputy Principal Medical Officer, and Dr. Hutton explored the liver with an aspirating needle and obtained a quantity of blood-stained serum. On the morning of the 1st of April I saw the case with Dr. Hutton and we decided to explore the liver again. On the seventh puncture, in the mid-axillary line below the fourth rib, a few shreds and a small quantity of pus appeared in the syringe. Cantlie's trocar and cannula was introduced, and the abscess-cavity was drained by a rubber tube dipping into carbolic lotion. Very little pus drained away, and the patient never rallied from the operation, and died the same evening with all the symptoms of acute toxæmia.

At the autopsy a large abscess-cavity, with ragged irregular walls, was found occupying the major part of the right lobe of the liver. Many of the exploratory punctures were traced into this cavity, which had evidently been filled by the fluid obtained at the first exploration. This lesion is typical of these severe toxic cases. The abscess-cavity is large, irregular in shape, and possesses no definite wall or lining membrane. The contents do not resemble the usual liver-pus, but consist of blood-stained fluid containing red corpuscles and granular debris. The infection is so acute that the process of liquefaction necrosis goes on much more rapidly than the inflammatory reaction in the liver. The cavity extends with great rapidity and may even increase in size after opening and drainage. A careful examination of the abdominal contents failed to reveal any trace of a primary focus for the hepatic abscess, and in particular no trace of dysenteric infection could be found in the intestine. The patient had been in the Colony only four months, and had had no previous tropical experience. No history of prior illness, particularly diarrhœa, was elicited, the deceased having been perfectly healthy till the onset of the fatal illness.

This case is interesting from several points of view. The short period of residence in the Colony and the absence of antecedent dysentery or diarrhœa are noteworthy. In the majority of cases a history of at least an attack of diarrhœa can be elicited, and the cases occur in people who, as a general rule, have been some considerable time in the tropics. Although the symptom complex was typical of liver-abscess, yet exploration of the liver by means of an aspirating needle of large calibre failed at first to reveal the abscess-cavity. In these acute toxic cases only blood-stained fluid may be obtained, and the absence of the typical liver-pus may give rise to doubt in the operator's mind as to the diagnosis. I know of one instance, in which the surgeon thought that a vein had been tapped, and subsequently an acute liver-abscess was found. The occurrence of such bloody fluid instead of the usual chocolate pus is to be borne in mind, and its appearance, combined with a leucocytosis, particularly of the polymorphonuclear variety, and a high remittent temperature, is of bad prognostic significance. Such cases frequently die from toxæmia, even where the abscess is found and drained. Herrick * reports two deaths in five cases.

Such cases serve to emphasize the unreliability of the aspirating syringe as a means of diagnosis. In the case quoted above many of the punctures could be traced into the abscess-cavity, yet the presence of a large cavity was not revealed. The diagnosis by means of the aspirating needle is of no negative value. In all cases where a reasonable suspicion is entertained of the presence of an abscess of the liver, even where repeated negative aspirations have been performed, it is better to perform a laparotomy in order to settle the question. An exploratory laparotomy is less dangerous than leaving an abscess, particularly of the severe toxic type. Moreover, there may be multiple abscesses present, and although one of these may be got by means of the needle the others may be overlooked. I have in mind a case where two abscesses were found by means of the needle and the patient died of a third which could not be located. It may happen that the abscess is in the left lobe of the liver and cannot be located by the aspirating needle. If a laparotomy is done, the whole liver can be carefully examined and, if need be, carefully and thoroughly explored.

With regard to operation, the trocar and cannula method of Manson as modified by Cantlie may in many instances be the method of choice. Particularly is it the case when the surgeon is alone and has not the resources of a properly equipped hospital at hand. The operation is speedy, free from great risk, and satisfactory where only one abscess-cavity exists which can be easily drained. But the abscess may fail to drain well, or there may be more than one abscess present, or as shown above the symptom complex may be diagnostic of liver-abscess and the aspirating needle has failed to locate its presence. In all such cases it is advisable to do a laparotomy ; such procedure is easier, speedier, and as safe as a transthoracic operation. I have employed the usual gall-bladder incision in the upper part of the right rectus, the abdominal cavity is packed off, and after the abscess is located a pair of sinus forceps is thrust into it and the pus evacuated. A large drainage-tube wrapped in gauze is passed to the bottom of the cavity and the wound closed in the usual manner. Hæmorrhage from the congested liver may be troublesome, and the cavity may require to be packed for the first twenty-four hours and the drainage-tube inserted subsequently.

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* A. B. Herrick, Surgery, Gynecology, and Obstetrics, Nov. 1910.

TRYPANOSOMIASIS. LONDON SCHOOL OF TROPICAL MEDICINE. (3rd Report.)

By H. B. NEWHAM, M.R.C.S., L.R.C.P., D.P.H.

In our last issue appeared a second report on cases of Trypanosomiasis in England, mainly at the London School of Tropical Medicine, and reference was therein made to case No. 16, W. G., who had been under treatment since October 1909, and whose health remained fairly good in spite of the occasional appearance of trypanosomes in the peripheral blood.

At the end of October 1912, patient began to complain of weakness in the legs, rendering walking somewhat difficult. This weakness gradually increased till he could only walk with great difficulty and with a shuffling gait. More or less headache was complained of from time to time, but no marked tremor of the tongue or hands were noted. On examination of the legs a good deal of loss of muscular power was found, but no loss of sensation. The calf-muscles on compression were found to be somewhat tender. The knee-jerks were exaggerated, but no ankle clonus was made out. The treatment was by intramuscular injections of Atoxyl. He refused to allow any more intravenous injections of Antimony or other drugs. The periodical recurrences of pyrexia, which were first pointed out by Sir Ronald Ross in the Rhodesian case at Liverpool, were marked in the later stages, though not during the earlier part of the course of the disease. Usually during the pyrexial periods there was an increased number of trypanosomes in the blood.

At 9 A.M. on the morning of December 14th, 1912, the patient, following on a somewhat restless night with a good deal of headache, suddenly had an epileptiform seizure. The face was cyanosed and the eyes protruding. There was blood-stained froth on the lips, owing to the patient having bitten his tongue before the mouth could be opened. Breathing was laboured and stertorous. Marked convulsive movements rapidly set in, chiefly on the right side of the body. The right pupil was much dilated and the corneal reflexes lost on both sides. The deep reflexes were exaggerated, especially on the right side of the body.

After about half an hour the convulsive movements became less violent and the breathing got easier, and it was thought that patient was about to regain consciousness, but shortly after all the symptoms recurred as before. These convulsive attacks, with short remissions, recurred during the whole day up to the evening. Shortly after the beginning of the first convulsion the patient began to perspire freely, and this continued up to the time of death.

During the evening the twitching of the limbs ceased and rigidity passed off. The whole of the right side became flaccid, and as far as could be made out there was almost complete paralysis of the limbs. There was also paralysis of the right side of the face, and of the mouth and tongue, with ptosis of the right eyelid. In the course of the day, patient passed urine and fæces several times into the bed, and on three occasions vomited dark and offensive material.

During the night the breathing became much weaker, and on one occasion Cheyne Stokes respiration was noticed.

The following morning the breathing was very shallow, entirely costal in character, and the pulse was almost imperceptible. The pupils were both widely dilated. At 11 A.M. the pulse could not be detected at the wrist, and breathing was practically gone, death taking place at 11.30 A.M.

During the initial convulsive attack the blood was examined for trypanosomes, but none were found.

From the initial epileptiform attack up to the time of death the temperature ranged between 105° F. and 106° F.

Cerebro-spinal fluid was drawn off some 5 hours after death and examined, but no parasites were found, and no excess of leucocytes was detected. The fluid was quite clear.

The post-mortem examination performed some 30 hours after death revealed the following condition:—

Brain.—Weight 49 ozs. Dura mater not adherent to skull-cap.

On opening dura the pia mater was found injected and adherent over the right parietal region over a small area about the size of a shilling. Ventricles not dilated. No excess of fluid in same.

Lungs.—Left 17 ozs.

Right 23 ozs.

No adhesions of the left lung.

Hypostatic congestion of the base.

A few adhesions (fairly recent) at base and also at apex of right lung. Also recent interlobar adhesions between upper and middle lobes.

Hypostatic congestion at base.

Heart.—10 ozs. Somewhat flabby. Valves normal. No dilatation of the organ.

Liver.—70 ozs., with somewhat pale areas, ill-defined in the upper part and looking somewhat fatty.

Spleen.—25 ozs. Large white patch on superior external surface. Smooth and shiny, looking like well-organised fibrous tissue. Also smaller patch just below first. No external adhesions. No evidence of infarcts. White patches evidently due to chronic diffuse perisplenitis.

Kidneys.—Left 3½ ozs.

Right 4 ozs.

Both kidneys appeared quite healthy and capsule stripped readily.

Intestines.—Normal. No worms present. Well-marked Meckel's diverticulum present.

Smears made from all organs showed no trypanosomes, the post-mortem being made about 30 hours after death.

Microscopical Examination of Tissues.

Brain.—No changes noted in the cerebellum. In the cerebrum there was well-marked perivascular infiltration with small round cells.

Spinal cord.—Some slight perivascular infiltration similar to that seen in the brain, but less extensive, was noted in the grey matter of the cord in the cervical, dorsal, and lumbar regions.

Spleen.—Some small-celled infiltration of the organ with well-marked and fairly extensive patches of congestion.

Kidney.—Slight interstitial fibrosis. Some cloudy swelling of the cells.

Liver.—Slight fatty degeneration of the cells, chiefly affecting those at the periphery of the lobules.

Testis.—Nothing abnormal detected.

This is the third case of Rhodesian Trypanosomiasis which we have had under observation. The other two died 5 months after infection from Trypanosomiasis, but without development of cerebral symptoms. This patient lived for 3 years and 3 months, and treatment was commenced within less than a month of the onset of symptoms. In spite of this, the disease was never more than under control, and even if the antimony injections had been permitted the lesions found indicate that the fatal termination would only have been postponed.

The contrast between the three cases of Rhodesian Trypanosomiasis, all fatal from the disease itself, and the ten cases from other parts of Africa, with two deaths from intercurrent diseases, indicates the greater malignancy of the Rhodesian infection. The treatment was on similar lines, though milder and less protracted in the other cases.

Case of Guinea Worm.

By D. J. F. O'DONOGHUE, Capt. R.A.M.C. (Gold Coast.)

Patient, a native policeman, reported sick on the 18th of January, complaining of headache and vomiting, pain and swelling of the right leg. Temperature 102° F.

Blood-examination.

	Differential count. Per cent.
Malarial Parasites	Nil.
Polymorphonuclears	57
Lymphocytes	18
Large Mononuclears	10
Eosinophiles	15

On examination a fluctuating tumour was found over the middle half of the right tibia, and it was noticed that the leg from the hip to the ankle was covered with an urticarial rash. On making an incision into the abscess a quantity of reddish-looking pus escaped and with it a bunch of Guinea Worms. On washing away the pus, it was found that there was one long female Guinea Worm and two small ones coiled round and adherent to her, and a third small one lying free at the bottom of the abscess-cavity. On pulling with a dressing forceps both the small specimens were removed, but the large one was fixed by its tail end deeply in the tissues, and was gradually removed by winding round a match. The patient informed me he never had a Guinea Worm before, and on looking up his Medical History sheet I find his statement correct. He also states that he got his drinking-water from the Goat Tank, and that he never drank any other water. I have repeatedly examined the water of this tank for the past 6 months and never found any Cyclops.

Dr. H. Werner on Cases of Human Trypanosomiasis caused by *T. rhodesiense*, with special reference to the Results of Lumbar Puncture.

[Ueber menschliche Trypanosomiasis mit Schlafkrankheitssymptomen aus Portuguesisch-Ostafrika, verursacht durch *Trypanosoma rhodesiense*, und über lumbalpunktatsbefunde, insbesondere die Nonne-Apeltsche. Phase 1. Reaktion bei Schlafkrankheit. By Dr. H. Werner.]

The paper deals especially with a case of Human Trypanosomiasis, the early history of which was described by Dr. H. Stannus in the 'Sleeping Sickness Bulletin,' 1912, vol. 4, n. 39, p. 266.

The patient (A. A. E.) was under treatment in the Albert Dock Hospital for about six weeks, and notes of the case were published by Dr. Daniels in the last number of the 'Journal of the London School of Tropical Medicine' (vol. 2, part 1, p. 92, A. A. E.). The patient left the Albert Dock Hospital, and was admitted to the Seamen's Hospital in Hamburg on Sept. 21st, 1912, where he remained till his death on October 6th. His condition in the Hamburg Institute, described by Dr. Werner in the paper under review, corresponds with Dr. Daniels' account of the case. Towards the end of the illness the patient exhibited a marked desire for sleep, a feature which appears to be much less frequently present in the cases due to *T. rhodesiense* than in those due to *T. gambiense*. The general weakness and œdema of the lower limbs increased from day to day. The urine had to be drawn off with a catheter. Further, Dr. Werner noted a rapidly increasing dimness of vision, which on September 28th, on the fourth day after the last injection of ·5 gram atoxyl, terminated in almost complete blindness. The patient had received two doses of half a gram of atoxyl on September 24th and 25th. He also received some intravenous injections of antimony tartarate in doses of 0·15–0·29 grams. On October 4th an examination of the left eye showed marked atrophy of the optic nerve. It was impossible to examine the other eye on account of the movements of the patient. There was considerable mental dullness, and during the last week there occurred a rapid increase in size of the lymphatic glands—in the left groin they increased to a tumour the size of a chicken's egg. On October 4th lumbar puncture was performed. The fluid was quite clear like water and trypanosomes were readily found in it without centrifugation. The Phase I. reaction (Nonne) was positive. The lymphocyte reaction was negative, while the Wassermann reaction was negative with the lumbar spinal fluid, though with the peripheral blood it had been positive.

The trypanosomes had proved themselves resistent to both atyoxl and antimony tartarate, and the numbers in the peripheral blood had remained high (up to 86 per c.cm.). However, during the course of October 5th the trypanosomes gradually diminished, so that 18 hours before the patient's death the trypanosomes had completely vanished—a result, according to the author, of an overloading of the blood with carbonic acid in the last stages of disease.

At the post-mortem examination the spleen was found enlarged (22 × 16 × 6 cm.), and the liver slightly so. Histological examination of the organs was not completed at the time the paper was written. C. M. W.

THE RESULTS OBTAINED IN A FEW CASES OF BUBONIC PLAGUE BY THE INTRAVENOUS INJECTIONS OF DILUTE SOLUTION OF IODINE.

BY F. POWELL CONNOR, F.R.C.S. (Eng.), Capt. I.M.S.

THE following notes on the treatment of bubonic plague are of some interest. The method suggested was only tried in three cases, and therefore it is in no sense convincing. The only object I have in publishing the facts is to enable others, if they think fit, to give it a further trial. The three cases were treated in the Gaya District in India, during the winter of 1910–1911. Scattered epidemics occur in this district each winter season. I had no opportunity of testing the method on other cases.

I. A native police constable was admitted into Hospital suffering from plague. He was semi-comatose, temperature 104°, and had a large and evidently tender right femoral bubo. Plague bacilli were found in a smear from the peripheral blood and in the fluid aspirated in a hypodermic needle from the lymphatic glands. My experience of plague convinced me that no ordinary method of treatment would save the man's life. I accordingly determined to inject a dilute solution of Iodine in small doses intravenously. An ordinary hypodermic needle was filled with a solution of Tinct. Iodi & Aq. Destill. (Tinct. Iodi ♏ vii & Aq. Destill. ʒi), and ʒi of the solution was injected into one of the veins of the forearm. The injection was repeated twice the same night.

The patient was better on the following morning, but was still dangerously ill. The temperature had fallen and the man was more conscious. In the course of the day, three more injections of the same strength were given intravenously. The same evening the temperature was about 100° and the patient's condition had obviously improved, and on the following morning he was quite conscious; he had but little fever, but was very weak. After this his recovery was slow, but uninterrupted.

II. A Mahomedan boy, aged about 10 years, the son of the Sub-Inspector of Police at the Gaya Police Station, was seen by me; there was a femoral bubo and the symptoms of an ordinary attack of bubonic plague were found. He was injected twice daily intravenously with Tinct. Iodi solution (♏v ad ʒi). After 3 days his condition was much improved, and recovery slowly followed. After the attack he was very weak with some dilatation of the heart, but he made a perfect recovery.

III. The third case was a boy of about 12 years of age, the brother of the last patient. He contracted plague 3 or 4 days after his brother, and was treated in the same way. The case developed and terminated without any untoward incident.

These three cases are reported for what they are worth, as no claims can be made with regard to this method of treatment on such insufficient evidence. The fact, at any rate, is obvious, viz., that the intravenous injection of Iodine in these cases had no prejudicial effect as far as could be ascertained. The rapid recovery of the first patient, who was suffering from a usually fatal form of plague, was very striking to me. I have seen some hundreds of cases of plague, but have no recollection of a patient in a similar condition ever recovering.

I have no personal knowledge of the use of injections of Iodine in other cases for plague or any other disease. But I have been able to ascertain (through the kindness of Sir Lauder Brunton, Bart., F.R.S.) that experiments with *large* doses of Iodine, with direct injection into the blood, were made by Boehm and Berg, and resulted in œdema of the lung and much hæmorrhagic exudation into the pleura and great congestion of the kidneys. Davaine found that when added to septicæmic blood it destroyed its infective quality, in the proportion of one in ten thousand, and somewhat similar results were obtained by Krajewsky with the blood of anthrax.

It is hard to believe that rather less than ʒi of Tinct. Iodi injected in small doses intravenously, as was done in the first case, is sufficient to disinfect the whole body in a severe infection of plague, but, according to Davaine and Krajewsky, this amount is probably sufficient to destroy the infective quality of the whole mass of circulating blood. It is conceivable that what remains of the infection can be dealt with by the natural resistance of the tissues.

The treatment was tried by me on purely empirical grounds, and the results in the few cases treated were certainly striking, but the actual value of the method can only be determined by further work on the same lines. It is tempting to speculate on the result of such treatment in the case of other diseases, in which the bacterial or protozoal infection is present for the most part in the circulating blood. Certain organic compounds of Iodine are normally present in some tissues of the body, and it is possible that small doses of Iodine, sufficiently strong to be of antiseptic value, may still be tolerated.

NOTICES OF BOOKS.

Aids to Tropical Hygiene. By Major R. J. BLACKHAM, R.A.M.C.
London: Baillière, Tindall, & Cox, 1912. 3*s.* net.

THIS book consists of 192 pages, including Index and two Appendices. In it the author gives a clear and concise account of the essential outlines of the subject, and the work should prove of some value to those wishing to revise their knowledge. Two chapters "Insects and Disease" and "Animal Parasites" take up 45 pages. These subjects form in reality all that part of Tropical Hygiene which differs markedly from the hygiene of temperate regions; and the aid would perhaps have been the greater for a fuller treatment, even within the limits of the book set by its title, of this branch of Tropical Sanitary Science. A useful chapter is devoted to "Prevention of Malaria." The Appendices comprise a table of tropical diseases indicating preventive measures, and one showing the distinctive characters of the more important species of flies. The list of references includes some of the better-known English text-books and treatises on Tropical Medicine, Sanitation, and kindred subjects. In the Preface Major Blackham quotes from Meredith Townsend's 'Asia and Europe':—"It is probably much more possible for white men to colonize a tropical country than is imagined, especially if the Colony was so organized that sanitary laws could be enforced from the very first." One may point out that to-day "certainly" could be written instead of "probably," for "the enforcement of sanitary laws from the very first" has actually been done—in the Panama Canal Zone—with results wonderful and so constant that nobody now wonders at all. H. M. H.

The Care and Treatment of European Children in the Tropics. By MONTAGU HARSTON, M.D., &c., Ophthalmic Surgeon to the Tungwa Hospital, Hongkong; and Examiner in Materia Medica and Therapeutics, Hongkong College of Medicine. With an Introduction by Sir PATRICK MANSON, G.C.M.G., M.D., LL.D. London: Baillière, Tindall, & Cox. 7*s.* 6*d.* net.

DR. HARSTON writes in the Preface: "The practitioner who proceeds early in his career to the tropics has nowadays generally had the advantage of having passed through a course at one of the Schools of Tropical Medicine. Such a course is necessarily mainly devoted to tropical diseases as they concern adults. After arrival in the tropics the practitioner may therefore find himself confronted with some practical problem intimately related to children, for the elucidation of which he will consult in vain the various treatises on children's diseases which he may have in his possession. Such a disease as malaria is, in some, not even mentioned, and dysentery is generally dismissed in a few lines. . . . . ."

The author has made time during the midst of a busy practice in Hongkong to give in book form the results of 15 years' experience in the treatment of children in the tropics, in the hope that it might be of some assistance to newly-arrived practitioners.

Sir Patrick Manson, in his Introduction to this book, writes: "The care of the European child in the tropics is yearly becoming of increasing importance. As Dr. Harston points out, whereas formerly few Europeans in the tropics were married, or, if married, allowed their children to remain for any length of time under tropic skies: nowadays it is otherwise, and the management of the health of the European child has become a serious part of the tropical practitioner's daily duties. . . . . ."

After reading the book the reviewer can but quote further, with appreciation, from this Introduction: "For teaching purposes in tropical medicine . . . . . . a book can never adequately take the place of the laboratory or of the hospital ward. Some books, however, by their freshness, clearness of expression, and avoidance of unnecessary detail or discussion, go a long way to make up for the absence of such advantages. Dr. Harston's is such a book . . . . . it will convey many useful hints, especially in treatment—a subject on which Dr. Harston's wide experience enables him to write with authority."

The interest and value of the book, one must add, are increased by Dr. J. Bell's excellent microphotographs of helminthic parasites and their ova.

H. M. H.

I.—ENTOMOLOGICAL NOTES.

Synopsis of the Anopheline Mosquitoes of Africa and of the Oriental Region.

By Colonel A. ALCOCK, C.I.E., F.R.S., I.M.S. (retired).

This Synopsis is intended for the convenience of medical men, my experience at the School having taught me that the method of identifying Anopheline species by attending, first and foremost, to the exact form and distribution of the scales of the body is—other grounds of criticism apart—confusing and sometimes misleading.

I would by no means ignore "scale characters," since I think that the nature of the scales, taken with the general scheme of colouring of the wings, provide the means of partition of the old genus *Anopheles* into four natural and fairly convenient subgenera, namely *Anopheles* (s. r.), *Myzorhynchus*, *Myzomyia*, and *Nyssorhynchus*.

I. Synopsis of African Species of Anopheles, ♀.

(*a*) *Wings not spotted at all* :—

1. Anopheles algeriensis, Theob. A brown mosquito without any banding of palpi or legs. Abdomen and scutum with hairs only. N. Africa. (Possibly identical with *A. bifurcatus*, L.)

(*b*) *Wings with some minute pale spots on the front edge only* :—

2. Anopheles smithii, Theob. Wings with 3 minute and obscure pale specks along the front edge and a fourth at the tip, otherwise brown-scaled. Palpi slender, dusky, with 3 narrow and obscure pale bands, one of which is subterminal. Legs brown, not banded. Abdomen and scutum with hairs only. Sierra Leone.

3. Anopheles rhodesiensis, Theob. As *smithi*, but the specks on the front edge and tip of the wing and the bands on the palpi are more distinct. Southern Africa.

(*c*) *Wings with streaks or spots on the front edge and fringe only* :—

4. Anopheles wellcomei, Theob. The front edge of the wing shows as a well-defined black line with 2 yellow breaks in its distal half; the fringe is spotted; the rest of the wing is covered with *yellow* scales. Palpi slender,

the proximal third black, the distal two-thirds light brown, with one narrow and two broader pale bands. Legs brown with the tips of the segments yellowish but not showing as distinct bands. Abdomen and scutum with hairs only. Sudan (and Aden).

5. ANOPHELES THEILERI, Edw. (= *Pyretophorus albipes*, Theob.). As *wellcomei*, but in the hind legs the 4th and 5th tarsal segments and the greater part of the 3rd also are white. Transvaal.

(*d*) *Wings with a few black spots in the field only, none on front edge or fringe* :—

6. ANOPHELES MACULIPENNIS, Meigen. Wings clad with narrow brown scales, which in four places are massed to form large black spots. Legs and palpi brown, not banded. Abdomen and scutum with scales only. A large brown species. N. Africa.

(*e*) *Wings dappled* ; *front edge* (*including tip*) *with not more than two pale spots* (fig. A) :—

7. ANOPHELES UMBROSUS, Theob. (= *Myzorhynchus umbrosus* and *strachani*, Theob.). A large dark species, with rather shaggy black palps. Wings with broad scales mostly black, but with patches of white; front edge black with two very small whitish spots in the distal half—the proximal one sometimes wanting. Legs dark brown, the extreme tips of most of the segments lighter. Abdomen and scutum with hairs only. W. Africa (also Malay Peninsula and Borneo).

8. ANOPHELES DISTINCTUS, Newstead & Carter. (*Pyretophorus distinctus*). Palpi slender with four narrow pale bands. Scutum with narrow scales. N.E. Rhodesia.

9. ANOPHELES MAURITIANUS, Grandpré (= *Myzorhynchus mauritianus* and *paludis*, Theob.). Palpi shaggy, black with usually four narrow white bands. Wings with broad scales, mostly black but with patches of white or yellow; front edge black with a small white spot at the tip, and usually another minute spot just beyond the middle. In the hind-legs the 3rd, 4th, and 5th tarsal segments are snow-white (sometimes the proximal end of the 3rd segment is dark = var. *paludis*). Abdomen and scutum with hairs except for a prominent tuft of scales on the sternum of the 7th abdominal segment. Widely distributed in Africa.

10. ANOPHELES AUSTENI, Theob. (*Pyretophorus austeni*). Palpi slender with 3 white bands, namely 1 narrow (proximal) and 2 broad—one of the latter being terminal. Front edge of wing black with 1 or 2 white specks near the base and 2 small white spots in the distal half. All the tarsal segments except the 5th have a white tip. Scutum with narrow scales. Angola.

(*f*) *Wings much spotted; front edge (including tip) with at least three distinct spots or streaks, but usually broken into numerous alternate light and dark areas* (figs. C, D). *No scales on the abdomen, or, at most, a few on the terminal segments*:—

(*fi*) *Palpi not banded*:—

11. ANOPHELES NILI, Theob. (*Myzomyia nili*). Palpi slender, dark with only the tip pallid. Wings dark-scaled with a few small light spots; the front edge black with four small whitish spots—one, at the tip of the wing, being a mere speck. Legs blackish, not banded. Abdomen and scutum with hairs only. Sudan; W. Africa.

(*f* ii) *Palpi with three light bands. Legs brown, the extreme tip of femur, tibia, and first* 3 *or* 4 *tarsal segments may be pale, but there is no distinct and conspicuous tarsal banding* [No. 12 to No. 20]:—

In the three following species (No. 12 to No. 14) *the tip of the palpi is brown*:—

12. ANOPHELES HISPANIOLA, Theob. (*Myzomyia hispaniola*). Palpi slender, brown, with three narrow light bands, the most anterior *not* being terminal. Legs brown with the tip of femur and tibia pale yellow. Front edge of wing black with four yellowish breaks, not including one or two specks at proximal end. Scales of 3rd longitudinal vein mostly pale. Abdomen and scutum with hairs only. N. Africa (and Spain). Said to rest like a *Culex*.

13. ANOPHELES CHAUDOYEI, Theob. (= *Pyretophorus chaudoyei* and *nigrifasciatus*, Theob.). Differs from *A. hispaniola* in having distinct scales on the scutum. N. Africa (also Cyprus and India).

14. ANOPHELES MULTICOLOR, Camboulin. Differs from *A. hispaniola* in having scales on the scutum, and from *A. chaudoyei* in having chiefly dark scales on the forks of the 2nd longitudinal vein. Egypt.

In the six following species (No. 15 to No. 20) *the third palp-band is terminal* (*tip of palpi white*):—

15. ANOPHELES CULICIFACIES, Giles (= *Myzomyia culicifacies* and *Pyretophorus sergenti*, Theob.). Palpi slender, brown, with three narrow light bands, one of which is terminal. Legs brown, with tip of femur and tibia and, perhaps, of some of the tarsi paler. Front edge of wing black with four small yellow breaks. Abdomen and scutum with hairs. An Indian species which has been found in Algeria. Resting attitude said to be Culex-like. N. Africa (a common Indian species also).

16. ANOPHELES FUNESTUS, Giles (*Myzomyia funesta*, Theob.). Differs from *A. culicifacies* in its darker appearance, the legs being almost black throughout. Resting attitude said to be not at all Culex-like. A common species in Tropical Africa.

17. ANOPHELES MARSHALLI, Theob. (= *Pyretophorus marshalli* and *pseudocostalis*, Theob.). Palpi slender, brown with three light bands—two broad (one of these terminal) and one narrow. Legs deep brown, the extreme tip of all the segments (except the 5th tarsal) pallid, but no distinct banding. Front edge of wing black with four small yellowish spots, not including 2 specks at proximal end. Scutum with narrow curved scales; abdomen with hairs. Eastern and Southern Africa.

18. ANOPHELES TRANSVAALENSIS, Carter (*Pyretophorus transvaalensis*). Seems to differ from *A. marshalli* chiefly in having darker legs. E. and S. Africa.

19. ANOPHELES FLAVICOSTA, Edw. Differs from *A. marshalli* in having more yellow in the front edge of the wing, which may be described as being yellow with 2 black specks and 4 small black spots. N. Nigeria.

20. ANOPHELES BRUNNIPES, Theob. (*Nyssorhynchus brunnipes*). Scutum with broad, elliptical, appressed white scales. Palpi slender, black with three white bands—two narrow, and one broad (terminal). In the legs some of the segments are smudgy black or brown and others almost golden yellow, but there is no definite banding. Front edge of wing black with 4 small white spots, not including a basal speck. Angola. This species is closely related to *A. rufipes* (No. 30), *A. maculipalpis* (No. 29), &c.

(*f* iii) *Palpi with three or four light bands, one of which is terminal. Some at least of the legs with tarsal segments distinctly and conspicuously banded* [No. 21 to No. 25]:—

[*Anopheles austeni*, Theob. (*Pyretophorus austeni*). See No. 10. In the front edge of the wing there are 3 white spots, but one of them is a mere speck.]

21. ANOPHELES COSTALIS, Loew, Theob. (*Pyretophorus costalis*, Theob.). Palpi slender, with 2 narrow bands and 1 broad terminal band. Legs brown with femur, tibia, and to some extent the first tarsal segment finely speckled with yellow, and the articulations of the tarsal segments banded yellow particularly in the front legs. Front edge of wing with 2 black specks and 4 black streaks, some of which sometimes tend to be confluent. Scutum with narrow scales; abdomen with hairs. One of the commonest African species.

22. ANOPHELES LONGIPALPIS, Theob. (*Myzomyia longipalpis*). Palpi slender with 3 narrow bands, one of which is terminal. Legs black, not speckled, the articulations of the tarsi of the hind-legs only banded white. Front edge of wing black with 4 small pale yellow spots. Scutum with hairs and a few narrow scales: abdomen with hairs. Proboscis particularly long. E. Africa.

23. ANOPHELES PITCHFORDI, Giles. Palpi slender with 3 bands, 1 narrow and 2 broad, one of the latter terminal. Legs dark brown with the tips of all segments, except 5th tarsal, yellowish and showing as distinct tarsal "bands." Front edge of wing black, with 2 yellow specks and 4 small yellow breaks. Scutum with narrow scales; abdomen with hairs. Southern and Eastern Africa.

24. ANOPHELES CINEREUS, Theob. (*Pyretophorus cinereus*). Palpi slender with 4 bands, one of which is terminal. Legs black, with the tips of all segments except 5th tarsal whitish, the white tips of the tarsi of the hind-legs forming distinct bands. Front edge of wing black with 1 or 2 yellow specks and 4 yellow streaks. Scutum with broadish scales: abdomen with hairs. South and East Africa.

25. ANOPHELES ARDENSIS, Theob. (= *Pyretophorus ardensis* and *Myzomyia pyretophoroides*, Theob.). Palpi slender, brown, with 4 white bands, 3 of which are narrow and 1 (terminal) broadish. Legs dark brown, with femur, tibia, and 1st tarsal profusely and conspicuously banded and all the other tarsals (5th excepted) white-tipped. Front edge of wing black with 2 yellow specks and 4 yellow breaks. Abdomen and scutum with hairs. Natal.

(*f* iv) *Palpi with three or four white bands, one of which is terminal. Legs banded; in the hind pair the 4th and 5th tarsal segments, and all or part of the 3rd segment also, are snow-white* [No. 26 to No. 30]:—

N.B.—The same snow-white hind tarsi occur in *A. theileri* (No. 5), *A. mauritianus* (No. 9), and *A. implexus* (No. 31). No. 5 and No. 9 can be excluded by the colouring of the wings, and No. 31 by the laterally-projecting pencils of bristle-like scales of the abdominal segments.

26. ANOPHELES NATALENSIS, Hill & Haydon (*Myzorhynchus natalensis*). Palpi black, with 4 narrow white bands, one of which is terminal. Femora and tibiæ brilliantly spotted, and first tarsal segment much banded, with yellow. In the hind-legs about half the 3rd tarsal segment is white (in addition to the 4th and 5th). Front edge of wing black with a white speck and 3 small white breaks. Scutum with hairs. Natal.

N.B.—Edwards appears to suggest that this "species" may be the same as No. 27.

27. ANOPHELES AUREOSQUAMIGER, Theob. (*Pyretophorus aureosquamiger*). Palpi slender, black, with 4 narrow white bands, one of which is terminal. Long segments of the legs profusely spotted, and tarsi broadly banded, with white. In the hind legs all or the greater part of the 3rd tarsal segment is (in addition to the 4th and 5th segments) white. Front edge of wing black, with one speck and 4 small spots of yellowish white. Scutum with broad, elliptical, appressed scales. Transvaal.

28. ANOPHELES PRETORIENSIS, Theob. (*Nyssorhynchus pretoriensis*). Differs from *aureosquamiger* principally in having all the markings white instead of yellow; the tarsi of the middle legs not, or indistinctly, banded; and the palpi with only 3 bands (though occasionally the terminal band is subdivided). Transvaal, Natal.

29. ANOPHELES MACULIPALPIS, Giles (= *Nyssorhynchus maculipalpis* and *indensis*, Theob.). Like *pretoriensis*, but the palpi (in addition to the 3 white bands) are peppered with white scales; the profuse spotting of the femora and tibiæ is more regular; and in the hind-legs all of the 3rd tarsal segment is white. Africa, Mauritius (also occurs in India).

30. ANOPHELES RUFIPES, Gough (*Nyssorhynchus pretoriensis* var. *rufipes*, Gough, and *Anopheles watsoni*, Edwards). Like *pretoriensis* without the spotting of the femora and tibiæ. Tropical Africa; Transvaal.

N.B.—Nos. 27, 28, 29 and 30 form a series; No. 29 (*Anopheles maculipalpis*, Giles) was the first to be named, and Nos. 27, 28 and 30 might quite reasonably be regarded as varieties of *Anopheles maculipalpis*, Giles. *Anopheles brunneipes*, Th. (No. 20), though it has not the three terminal tarsi of the hind-legs snow-white, is very closely related to this series.

(*g*) *Wings as in* Section (*f*). *Legs profusely banded. Abdomen with a tuft of long stiff bristle-like scales projecting laterally on either side of each of the first seven segments* :—

31. ANOPHELES IMPLEXUS, Theob. (*Christya implexa*). Palpi rather shaggy, rather shorter than the proboscis, black with 4 or 5 narrow whitish cross-bands. Legs dark brown, the femur and tibia with numerous regular white cross-bands. In the hind-legs the tarsal segments from the middle of the 2nd to the middle of the 5th are snow-white. Wings spotted, two or three large yellow blotches on the anterior edge are very conspicuous. Scutum with broadish appressed scales among the hairs. Abdomen with scales on the terminal segment, and with a pair of laterally-projecting pencils of stiff narrow scales on the first seven segments. Uganda.

(*h*) *Wings as in* Section (*f*). *Palpi and tarsi banded. Abdomen well clad with scales on every segment* :—

32. ANOPHELES CHRISTYI, Newstead & Carter ("*Neocellia*" *Christyi*). Palpi shaggy, with three very narrow whitish bands and yellowish tip. Legs lightish brown, tips of all the tarsi except the 5th yellowish, forming distinct bands. Front edge of wing black, with 2 or 3 yellowish specks and 4 yellow breaks. Scutum with broadish curved scales. Plenty of scales on all the abdominal segments, those of the genital lobes being very prominent. E. Africa.

(*i*) *Wings as in* Section (*f*). *Legs profusely banded. Abdomen well covered with scales, and also with a laterally-projecting tuft of scales on either side of every segment from the 2nd to the 7th* :—

33. ANOPHELES SQUAMOSUS, Theob. ("*Cellia*" *squamosa*). Palpi shaggy, black peppered with white, and with 2 or 3 white bands and white tip, the banding variable. Legs with femur and tibia much speckled, and all the tarsi except the 5th (which is dark brown) broadly banded with white distally. Wings much spotted; front edge black with 2 white specks and 4 white spots. Scutum with broad elliptical appressed scales. A common species of the Ethiopian region.

34. ANOPHELES ARGENTEOLABATUS, Gough (="*Cellia*" *pseudosquamosa*, Newstead & Carter). Much like *A. squamosus*, but the tarsi are not banded. Transvaal & N.E. Rhodesia.

35. ANOPHELES PHAROENSIS, Theob. ("*Cellia*" *pharoensis*). Palpi much as in *squamosus*, and variable. Legs with femur and tibia brindled or irregularly banded, the tarsi (5th excepted) broadly banded in dark brown

and white. 5th tarsal segment of the hind-legs, only, white. Wings mostly yellow, with spots and blotches of dark brown; the front edge with 2 minute dark specks and 4 dark spots. Scutum with broad appressed scales, and with a pair of conspicuous laterally-placed dark brown spots. Common in Africa (also in Palestine).

36. ANOPHELES CINCTUS, Newstead & Carter ("*Cellia*" *cincta*). Said to differ from *A. pharoensis* in having the alternate banding of the tarsal segments particularly regular, and the 5th tarsal segment of all the legs yellow. Must be regarded as a very doubtful "species," as it is founded on a single female specimen (from Ashanti) with the palpi broken and the front legs wanting.

37. ANOPHELES JACOBI, Hill & Haydon ("*Cellia*" *jacobi*). Much like *A. pharoensis*, but the 5th tarsal segment of all the legs is white-tipped, and the other tarsal segments are not banded. Natal. This also seems to be doubtfully distinct from No. 35.

II Synopsis of Oriental Species of Anopheles, ♀.

(*a*) *Wings not spotted* :—

1. ANOPHELES AITKENI, James (= *A. treacheri*, Leicester, *Stethomyia fragilis*, Theob., and *Neostethopheles culiciformis*, James & Liston). Palpi and legs slender, not banded. Scutum and abdomen with hairs. Darts of head extremely slender. *Stethomyia pallida*, Ludlow, from the Philippines, is probably this species.

2. ANOPHELES BARIANENSIS, James. Differs from *A. aitkeni* in having normal darts on the head. Punjab Himalayas.

3. ANOPHELES IMMACULATUS, Theob. Palpi with 3 light bands, namely 2 narrow and a broad terminal. Legs yellowish, the tarsi darker, with faint terminal bands, least inconspicuous in hind-legs. Wings with yellowish veins and pallid scales. Scutum and abdomen with hairs. E. Coast of Madras.

(*b*) *Wings spotted. Femur of hind-legs with a conspicuous terminal ruff of outstanding scales not found in any other species* (except sometimes, according to C. Strickland, in *Anopheles barbirostris*) :—

4. ANOPHELES ASIATICUS, Theob. (= *Lophoscelomyia asiatica*). Palpi shaggy, not banded. The ruff on the hind femora consists of a proximal mass of outstanding black scales and a distal mass of outstanding white scales. Wing scales mostly dark coloured; two conspicuous yellow spots on front edge of wing. 8th abdominal segment covered with scales. Malay Peninsula.

5. ANOPHELES WELLINGTONIANUS, Alcock. The ruff on the hind femora consists entirely of outstanding black scales, proximal of which is a broad band of small *appressed* white scales. On the front edge of the wing there are two yellow spots, one of which (situated at tip) is conspicuous, the other (situated just beyond the middle) being a mere speck. No scales on 8th abdominal segment, but there may be a projecting wisp of scales on the 7th abdominal sternum. Malay Peninsula.

(c) *Wings dappled, the front edge black with only one, or two, light spots* (fig. A) ; *rather large species* :—

6. Anopheles lindesayi, Giles. Palpi slender, black, not banded. Legs black, a broad white band across middle third of hind femora much as in the nearly-related *A. wellingtonianus.* Wings spotted rather sparsely, the front edge black with one large white spot (at tip). Scutum and abdomen with hairs. Himalayas. A variety, *maculatus*, Theobald, from the Eastern Himalayas, has some additional black spots on the wing, arranged much as in *A. maculipennis.*

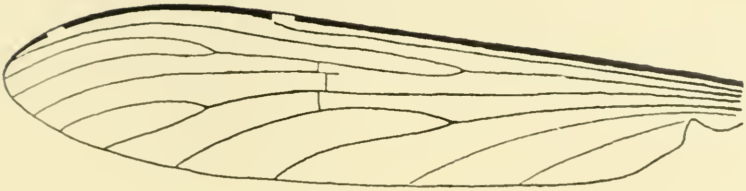
7. Anopheles umbrosus, Theob. (*Myzorhynchus umbrosus*). Palpi rather shaggy, black, not banded. Legs brown, the extreme tips of the tarsal segments paler, but not forming distinct bands. Wings mostly black scaled, the light spots few; the front edge black with a whitish spot at tip and usually a minute light spot just beyond the middle. Scutum and abdomen with hairs. Malay Peninsula and Borneo (also W. Africa).

8. Anopheles albotæniatus, Theob. (*Myzorhynchus albotæniatus*). Palpi shaggy, black, not banded. In the hind-legs the 5th tarsal segment is white, and there are white bands at the joints of all the other tarsal segments. Wings mostly black scaled, front edge black with two small yellow spots situated just as in *umbrosus.* Scutum and abdomen with hairs. Malay Peninsula.

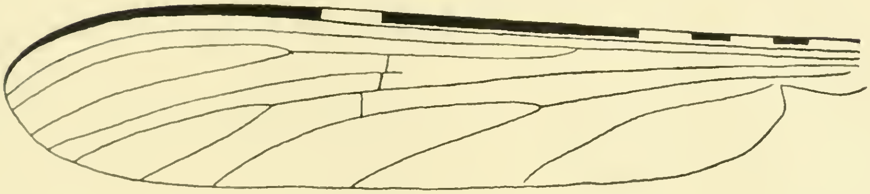
9. Anopheles barbirostris, v. d. Wulp (*Myzorhynchus barbirostris*, Theobald). Palpi very shaggy, black, not banded. Legs black with the tips of the segments pale, in the hind tarsi showing as distinct white banding. Wings mostly black scaled, the light spots few ; the front edge black with a whitish spot at tip and a minute and inconstant light spot beyond the middle. Scutum with hairs. Abdomen always with a projecting clump of dark scales on the 7th abdominal sternum, and usually with one or two projecting wisps of white scales on all or most of the other abdominal sterna ; occasionally the abdominal sterna are covered with scales. Oriental region generally.

10. Anopheles bancrofti, Giles (= *Myzorhynchus pseudobarbirostris*, Ludlow). Agrees with *A. barbirostris* in every particular, except that the femora and tibiæ are sparsely speckled with white. May reasonably be regarded as a variety of that species. Philippines (also Australia).

11. Anopheles sinensis, Wiedemann (= *Anopheles vanus*, Walker, *A. nigerrimus*, Giles, *A. pseudopictus*, Grassi, *Myzorhynchus minutus*, Theob., and *M. peditæniatus* and *separatus*, Leicester). Palpi rather shaggy, black, with 4 (sometimes 3, or even 2) narrow white bands, one of which is almost always terminal. Legs black, the tips of the segments light tawny and showing as distinct—though variable—tarsal banding. Wings black-scaled with some tawny patches ; the front edge black with a small and inconstant tawny spot beyond the middle and a larger one constantly near the tip. Scutum with hairs. A prominent tuft of scales on the 7th abdominal sternum. A common and variable Oriental species which is also found in the Palæarctic region, both to the north-east and north-west. *Anopheles plumiger*, Donitz, seems to be another synonym.



A



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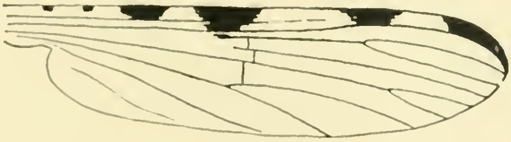
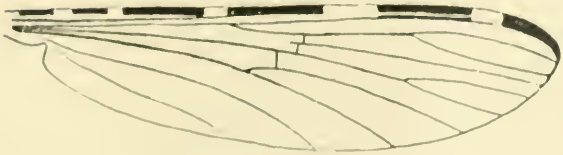


C



D

(*d*) *Wings spotted, the front edge in its proximal fourth white with two small black streaks, in its distal three-fourths black with one or two white streaks* (fig. B); *large species* :—

12. ANOPHELES GIGAS, Giles. Palpi slender, black, not banded, decidedly shorter than proboscis. Legs dark brown with lightish bands at the joints of all the segments. Wings mostly white-scaled with numerous black spots and streaks, two long and broad black streaks occupying the greater part of the anterior edge. Scutum and abdomen with hairs. Nilgheri Hills of S. India.

12*a*. ANOPHELES GIGAS, var. REFUTANS, nov. Differs from the typical form only in having 3 or 4 *very* narrow white bands on the palpi, one of them *usually* being terminal. Hills of Ceylon.

12*b*. ANOPHELES GIGAS, var. SIMLENSIS, James (*Patagiamyia simlensis*). Differs from the typical form only in having (*a*) 3 narrow white bands on the palpi, and (*b*) the bands across the inter-segmental joints of the legs broader and whiter. (The tip of the palpi is black, not white or yellowish as usually in var. *refutans*.) Western Himalayas.

13. ANOPHELES FORMOSUS, Ludlow. Palpi rather coarse, brown, with 3 narrow light bands, one of which is terminal. Legs brown, with yellowish knee-spots, and yellowish bands at the intertarsal articulations except the last of the front and middle legs. Wings yellowish with brown spots; front edge with 2 small and 2 large brown "spots." Scutum and abdomen with hairs. Philippine Is.

(*e*) *Wings spotted, the front edge broken into numerous alternate light and dark spots or streaks* (figs. C, D). *Palpi banded. Scutum never bearing broad appressed scales. Abdomen usually with hairs, but never bearing many or conspicuous scales* [No. 14 to No. 24] :—

(*e* i.) *Legs not distinctly banded*; *tip of palpi brown* :—

14. ANOPHELES TURKHUDI, Liston. Palpi slender with three narrow white bands and brown tip. Legs deep brown, the extreme tip of some of the segments may be paler, but there is no distinct tarsal banding. Wings dusky with yellow spots, the front edge with 2 yellow specks near base and 4 yellow spots or streaks. Scutum and abdomen with hairs. India.

15. ANOPHELES CHAUDOYEI, Theob. (= *Pyretophorus chaudoyei* and *nigrifasciatus*, Theob.). The vestiture of the scutum consists of very distinct scales, and the scales of the wings are more yellow; otherwise as *A. turkhudi*, Baluchistan (also N. Africa). James and Liston seem to have very well justified doubts whether this "species" is really distinct from *A. turkhudi*, which is the prior name.

(*e* ii.) *Legs not distinctly banded*; *tip of palpi white* :—

16. ANOPHELES CULICIFACIES, Giles (*Myzomyia culicifacies* in Theobald). Palpi slender, brown, with 3 white bands, one of which, a little broader than the others, is terminal. Legs brown, the extreme tip of some of the segments

may be paler, but there is no distinct tarsal banding. Wings with yellowish and tawny spots ; front edge dark with four almost equidistant and equal whitish streaks ; the 3rd longitudinal vein with mainly dark scales. Scutum and abdomen with hairs. Rests like a *Culex*. India and Ceylon.

Anopheles leptomeres, Theobald, is probably a synonym of this species.

17. ANOPHELES LISTONI, Liston (*Myzomyia christophersi*, Theob.). Differs from *A. culicifacies* only in having mainly yellow scales on the 3rd longitudinal vein and the light spots or streaks on the front edge of the wing smaller.

Anopheles minimus, Theob. (*Pyretophorus minimus*), from Hongkong, described from an imperfect female, seems from the description to be identical with *A. listoni* ; as also does *A. aconitus*, Dönitz.

(*e* iii.) *Some of the legs with distinct tarsal bands; tip of palpi white*:—

18. ANOPHELES ROSSI, Giles (*Myzomyia rossi*, Theobald, *Nyssomyzomyia rossi*, James & Liston). Palpi brown, with 3 white bands, two being narrow and the terminal one being broad. Legs brown, the long segments (5th tarsal excepted) all with a white tip, which—particularly in the case of the front legs—shows as distinct tarsal banding : under a lens the femora and tibiæ sometimes look speckled or mottled. Wings yellow with dark brown spots ; the front edge yellow with two fine dark brown specks proximally, and four dark brown streaks, the second of which has the shape of a squat **T**. Scutum with hairs. Some not very conspicuous narrow scales on the last two abdominal segments.

Very common throughout the Oriental region.

19. ANOPHELES LUDLOWI, Theob. (*Myzomyia ludlowi*, Theob., *Nyssomyzomyia ludlowi*, James & Liston). Differs from *A. rossi* chiefly in having the femora and tibiæ decidedly speckled, the terminal band of the palpi narrower, and the two narrow bands more widely separated. Most authorities regard it as a distinct species. But for the fact that it is said to be a malaria-carrier in the Andamans, I should regard it as a variety of *A. rossi*. Philippines, Malay Peninsula, India.

20. ANOPHELES INDEFINATUS (*Myzomyia indefinata*). Differs from *A. rossi* in having the terminal band of the palpi narrower, and the two narrow bands more widely separated. The differences between these three species—if they *are* distinct species—can only be appreciated by actual comparison of specimens. (The incidence of palp-bands is, in several species of *Anopheles,* so inconstant and variable, that it does not *by itself* supply a definition for a species.)

21. ANOPHELES JEYPORENSIS, James (*Pyretophorus jeyporensis*). Palpi slender, dark, with 3 white bands, two being narrow and the terminal one broad. Legs almost black, the extreme tips of the segments almost white, showing as distinct though very narrow tarsal banding. Wings with white and black spots and streaks ; the front edge black with two white specks and four white spots gradually increasing in size from the first to the third. Abdomen with hairs. Scutum with narrow white *scales*. Central and Southern India.

22. ANOPHELES ALBIROSTRIS, Theob. (*Myzomyia albirostris*). Basal half of proboscis black, distal half yellow. Palpi slender, the basal two-thirds dark, with a narrow, white, median band, the distal third yellow with (usually) a very narrow black median band. Legs dark brown, the tip of the first four tarsal segments light—forming distinct though narrow banding, at least in the front legs. Wings yellow with dusky spots, the front edge black with a yellow speck followed by three or four small yellow spots; all the scales of the 3rd longitudinal vein yellow. Malay Peninsula.

23. ANOPHELES PUNCTULATUS, Dönitz (=*Myzomyia tesellata*, Theob., and *Nyssomyzomyia punctulata*, James & Liston). Basal half and tip of proboscis black, distal half (tip excepted) light tawny or white. Palpi slender, basal half black with a narrow white band, distal half white with two very narrow dark bands (and so commonly described as having 4 white bands). Legs dark brown, the femur, tibia, and to a less extent the first tarsal segment regularly and copiously spotted light tawny, the next three tarsal segments with light tawny tips which (except in the hind-legs) involve the articulations, and in the front legs form broadish banding. Wings spotted, black and light tawny; front edge black with one or two tawny specks followed by four tawny spots. Scutum and abdomen with hairs. India, Malaya, Siam.

Anopheles thorntoni, Ludlow, from the Philippines, is probably this species.

Anopheles deceptor, Dönitz, from Sumatra, is so named on account of its deceptive (?) resemblance to *A. punctulatus* and *A. leucosphyrus*. From the description the principal difference from *A. punctulatus* appears to be in the details of the colouring of the palpi. These in the distal half are white with 2 dark rings, and in the proximal half are dark with the first segment white above. Such a difference cannot be regarded as specific.

24. ANOPHELES LEUCOSPHYRUS, Dönitz (=*Pyretophorus elegans*, Theob., and *Neomyzomyia elegans*, James). Proboscis black with white tip. Palpi slender with 4 white bands, the broadest of which is terminal. Legs as in *punctulatus*, but the spotting of femur &c. is white, and in the hind-legs the white band across the junction of the tibia and first tarsal segment is particularly broad. Wings much spotted; front edge black with 1 or 2 white specks followed by four white breaks. Scutum and abdomen with hairs. India, Ceylon, Sumatra.

(*f*) *Wings spotted, the front edge broken into numerous alternate light and dark spots or streaks* (fig. D). *Palpi and legs banded. Scutum with broad, elliptical, appressed scales which are usually white. There may be some scales on the abdomen, but never prominent lateral or sternal tufts*:—

25. ANOPHELES MACULATUS, Theob. (=*Nyssorhynchus maculatus* and *pseudowillmori*, Theob.). Basal two-thirds of palpi black with narrow white median band, distal third white with narrow black median band; *sometimes* there are also white scales sprinkled among the black. Proboscis black, pale at tip. Legs dark brown; femur, tibia, and first tarsal segment speckled with white or light tawny spots; in the front legs, and usually in the middle legs also, the articulations between the tarsal segments are white; in the hind legs the

2nd tarsal segment has a white tip, the 3rd is white with a broad dark band, the 4th is white with a narrower band, and the 5th is entirely white. Wings spotted black and white; the front edge black with 2 white specks followed by 4 white spots. Scutum with broad, white, appressed scales. Abdomen with some scales among the hairs on the last two segments. Northern India, Malay Peninsula.

25 *a*. ANOPHELES MACULATUS, var. WILLMORI, James (=*Neocellia willmori*, James, and *Neocellia dudgeoni* and *indica*, Theob.). Differs from the typical only in having scales on all the abdominal terga, and the black parts of the palpi not speckled as they sometimes are in typical *maculatus*. Punjab Himalayas, Kashmir and adjacent parts.

25 *b*. ANOPHELES MACULATUS, var. THEOBALDI, Giles. Like the type, except that in the hind-legs there is no dark band on the 4th tarsal segment, so that that segment is entirely white (as well as the 5th), and that the black parts of the palpi are not speckled. Peninsular India.

26. ANOPHELES FULIGINOSUS, Giles (=*Nyssorhynchus fuliginosus* and *nivipes*, Theob.). Palpi black with 3 white bands, two being narrow and one broad (terminal). Legs black or brown, the long joints not speckled; in the front and middle pair the apex of the first 3 tarsal segments is white; in the hind pair the last 3 tarsal segments and the distal end of the 2nd are snow-white. Wings spotted white on black; the front edge black with 4 or 5 small white spots. Scutum with broad, white, appressed scales. Abdomen with scales fairly plentiful on the last two terga. India in general; Malay Peninsula.

A. leucopus, Dönitz, seems to be a synonym of this species. *Calvertina lineata*, Ludlow, is nothing very different.

26 *a*. ANOPHELES FULIGINOSUS, var. PHILIPPINUS, Ludlow (*Nyssorhynchus philippinensis*, Ludlow). The broad terminal white band of the palpi is cut in two by a narrow band of dark scales, so that the palpi have four white bands. Philippines, Siam.

26 *b*. ANOPHELES FULIGINOSUS, var. ADIEI, James & Liston. The broad terminal white band of the palpi is cut in two by a narrow band of dark scales; in the hind-legs the 2nd tarsal segment and the proximal end of the 3rd are dark.

26 *c*. ANOPHELES FULIGINOSUS, var. FOWLERI (*Neocellia fowleri*, Christophers). The white spots on the front edge of the wing are larger, and scales may be present on several of the abdominal terga.

27. ANOPHELES JAMESI, Theob. (*Nyssorhynchus jamesi*). Differs from *A. fuliginosus*, Giles, principally in having the femur, tibia, and first tarsal segment of the legs copiously spotted with white. Peninsular India.

28. ANOPHELES MACULIPALPIS, Giles (= *Nyssorhynchus indensis*, Theob.). Differs from *A. jamesi* principally in having the palpi speckled with white (in addition to the 3 white bands). Peninsular India; N.W. Himalayas (also Africa).

The above, No. 26 to No. 28, form a very nice series, *A. maculipalpis*, Giles, having the right of priority. Compare the corresponding African series (p. 158), all probably referable to *A. maculipalpis*, Giles.

Anopheles freræ, Banks ("*Pyretophorus*" *freræ*) from the Philippines, would appear to come in the same series.

29. ANOPHELES STEPHENSI, Liston (= *Neocellia stephensi*, Liston, and *Neocellia intermedia*, Rothwell). Palpi dark, with two broad white bands (one being terminal) in the distal half, and two or three white patches or narrow bands in the proximal half. Wings spotted black and yellow; the front edge with 2 small black specks followed by four black streaks, the 2nd of which is squat T-shape as in *A. rossi*. Legs dark brown: femur, tibia, and to a less extent the first tarsal segment speckled with tawny spots, the tips of all the tarsal segments except the 5th light tawny. Scutum with appressed yellowish scales. Scattered scales on all the abdominal terga except the 1st. India generally.

Anopheles vagus, Dönitz, from the Malay Archipelago, though considered by the author to be closely related to *A. rossi*, appears from the figures and description to approach *A. stephensi*.

30. ANOPHELES KARWARI, James (= *Nyssorhynchus maculatus*, Theob. *partim*). Palpi dark with 4 white bands, namely, 2 narrow followed by 2 broad, one of the latter being terminal. Legs dark brown, not speckled; in the front and middle legs the tips of the first 3 tarsi are white, in the hind legs the tips of the first 4 and the whole of the 5th are white. Wings spotted black and white: front edge with 2 white specks and 4 white spots. Scutum with broad, appressed, white scales. Scales on last 2 abdominal segments. Bombay Presidency; Malay Peninsula.

(*g*) *Wings spotted; the front edge broken into numerous alternate dark and light spots or streaks* (fig. D). *Palpi and legs banded. Scutum with broad scales. Abdomen with numerous scales on all the segments, and also with regular tufts (lateral or ventral) of projecting scales*:—

31. ANOPHELES PULCHERRIMUS, Theobald (*Cellia pulcherrima*). Palpi brown frosted with white, and with 4 white bands, the broadest of which is terminal. Legs brown frosted with white, the tips of all the segments white; in the front and middle legs the 4th and 5th tarsi are brown, in the hind-legs the greater part of the 2nd and the whole of the 3rd, 4th, and 5th tarsi are white. Wings spotted brown on white; the front edge with 2 black specks followed by 4 black streaks. Scutum with broad, white appressed scales. Abdomen plentifully covered with broad white scales and with a pair of laterally-projecting tufts of dark scales on each segment. N.W. India, Persia, Bombay.

32. ANOPHELES KOCHI, Dönitz ("*Cellia*" *kochi*, Theob., *Christophersia halli*, James). Palpi described as displaying dorsally 5 broad white bands separated by 4 narrow black bands: they vary, even on opposite sides of the same individual: commonly they are dusky at base, and white and yellow in the distal three-fourths or more where there are 3 or 4 very narrow dark bands. In all the legs, the femur, tibia, and 1st tarsal segment are copiously brindled or banded: in the front legs both ends of the next 3 tarsal segments are white: in the hind-legs the distal end of the 2nd tarsal segment is white, and the next 3 segments are white with a dark band (gradually decreasing in breadth) across the middle. Wings spotted dark brown on yellow; front edge with numerous unequal, rather irregular, and not very large blackish spots. Scutum with broad white scales and narrow curved scales and hairs: a pair of dark round lateral spots are here conspicuous. Abdominal terga with hairs and scales: the sterna, from the 2nd to the 7th, each with a vertically-projecting bifid tuft of dark scales. Malay Peninsula and Islands; Assam.

33. ANOPHELES FLAVUS, Ludlow ("*Cellia*" *flava*) agrees—so far as description goes—with *A. kochi*, Dönitz, but the tufts of dark scales on the abdominal terga are stated to be *lateral*. Philippines.

EXPLANATION OF FIGURES.

Figs. A, B, C, D.—Diagrams of wing-patterns in the genus *Anopheles*.

SOME NOTES ON *STEGOMYIA FASCIATA* IN THE COAST TOWNS OF BRITISH SOMALILAND.

BY R. E. DRAKE-BROCKMAN, M.R.C.S., L.R.C.P.

(1) EGGS.

The eggs of *Stegomyia fasciata* are about 1–1·5 mm. in length, black or dark brown in colour and slightly curved. They are of an elongated oval shape with one end somewhat more obtuse than the other, while the surface has a rough and granular appearance.

They are always laid singly and never adherent together in rafts and usually along the water-line, and always within an inch of it adherent to the sides of the receptacle.

If the atmosphere is very dry and the water evaporates rapidly the eggs soon get left high and dry, but this does not seem to interfere with the majority of them except during the excessive heat of the summer months, when the maximum shade temperature rises as high as 109° F.

When the larvæ emerge the eggs are all usually fractured at about the same spot, obliquely where the outer third joins the inner at the obtuse end.

It is probable that all the eggs deposited in such receptacles as jam tins and in partly broken bottles after the rains and just previous to the hot summer, get destroyed owing to the rapid evaporation of the water and the heating of the receptacles, the species being continued by the individuals which æstivate.

(2) LARVÆ.

The larvæ emerge under favourable conditions in from 2–3 days after the eggs are deposited and pupate in about 4 or 5 days. If food is scarce the larval stage may last for three weeks or more.

They will thrive in the smallest quantity of water, a thimbleful being sufficient for half-a-dozen of them. In point of fact, where water is plentiful in any receptacle it is surprising how many larvæ will exist together and thrive without devouring each other, a not uncommon occurrence when the food supply is getting scarce.

Stegomyia larvæ will coexist in the same water with those of *Culex salus* and probably other larvæ.

They prefer rain-water although brackish well-water is also acceptable.

(3) PUPÆ.

The pupal stage lasts about 24 hours, sometimes more, sometimes less, and there is little doubt that large numbers of the pupæ die in nature as they do when bred under artificial conditions. The great heat, especially during the last fortnight in June, on the Somali coast seems to be too much for them, as I have been unable to continue breeding experiments after that date under natural conditions.

(4) IMAGO.

As soon as the perfect insect emerges from the pupal case, head first, a process which only takes a few minutes, it rests for a short time on the surface of the water until it is ready to feed; this is not before 12 hours have elapsed, as I have never got the females to come to the arm on the day of their birth.

It is at this stage that some of their non-aquatic enemies attack them, and the commonest of these is the common little red ant so ubiquitous in the Tropics.

Doubtless these same ants, together with spiders, destroy large numbers of the æstivating mosquitoes as well.

In Table I. will be found a number of experiments conducted with a view to ascertain the length of life of *Stegomyia fasciata* when unfed, fed on dates, and lastly on human blood. In Table II. will be found the time occupied by the different stages of its life-history.

EXPLANATION OF TABLE I.

Exp. 1. *Fed on dates.*

All the females were dead by Dec. 25th and males by Jan. 22nd.

Exp. 2. *Unfed.*

All the 29 mosquitoes, male and female, were dead by the fourth day.

Exp. 3. *Fed on blood.*

Of the five experimented with, the males all died before the third day. One female escaped, while the other died on Jan. 9th, after surviving eight days.

Table I.

Stegomyia fasciata.

Exp.	No.	Sex.	If Fed.	How Fed.	Date of Birth.	Date of Death.	Total length of Life.
1	9	♂ & ♀	Yes	Dates	18. 12. 11	♀ 25. 12. 11 ♂ 22. 1. 12	7 days 35 „
2	29	♂ & ♀	No	..	19. 12. 11	23. 12. 11	4 days
3	5	3 ♂, 2 ♀	Yes	Blood	1. 1. 12	3. 1. 12 9. 1. 12	2 days 8 „
4	60	♂ & ♀	No	..	7. 1. 12	10. 1. 12	3 days
5	16	♂ & ♀	Yes	Blood	8. 1. 12	27. 1. 12	19 days
6	12	♂ & ♀	No	..	5. 1. 12	9. 1. 12	4 days
7	7	♂ & ♀	No	..	9. 1. 12	11. 1. 12	2 days
8	6	4 ♂, 2 ♀	Yes	Blood	9. 1. 12	12. 1. 12	3 days
9	20	13 ♂, 7 ♀	Yes	Blood	28. 1. 12	2. 2. 12 2. 2 12 7. 2. 12 8. 2. 12 26. 2. 12	5 days (13 ♂) 5 „ (2 ♀) 10 „ (1 ♂) 11 „ (1 ♂) 30 „ (3 ♀)*
10	5	♂ & ♀	No	..	29. 1. 12	2. 2. 12	4 days
11	8	♂ & ♀	Yes	Dates	30. 1. 12	17. 2. 12	18 days
12	9	6 ♂, 3 ♀	Yes	Dates	20. 2. 12	22. 2. 12 25. 2. 12 26. 2. 12 1. 3. 12 14. 3. 12	2 days (1 ♂) 5 „ (2 ♂, 2 ♀) 6 „ (1 ♂) 10 „ (2 ♂) 22 „ (1)

Exp. 4. *Unfed.*

Out of 60 mosquitoes, male and female, none survived more than three days.

Exp. 5. *Fed on blood.*

On Jan. 10th only one male and three females were alive. Of the survivors the male died on Jan. 11th, one female on Jan. 15th, while the other died on Jan. 27th.

Exp. 6. *Unfed.*

All died after surviving four days.

Exp. 7. *Unfed.*

Only survived two days.

Exp. 8. *Fed on blood.*

Females refused to feed. All died in three days.

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* One escaped Feb. 20th, while the two surviving females were accidentally mixed with another brood.

Exp. 9. *Fed on blood.*

In this experiment there were 13 males and 7 females. Four days later only 2 males and 4 females were alive, while on Feb. 2nd both males and two of the females were dead. Previous to this, on Jan. 30th, they were seen in copula, and on Feb. 6th larvæ were hatched from the eggs. On Feb. 20th only two females were alive, one having escaped, while the two survivors were accidentally mixed with another brood and the experiment abandoned.

Exp. 10. *Unfed.*

Males and females survived four days.

Exp. 11. *Fed on dates.*

All dead by the eighteenth day.

Exp. 12. *Fed on dates.*

One male died on Feb. 22nd.
Two males and two females on Feb. 25th.
One female on Feb. 26th.
Two males on March 1st.
The last surviving male on March 14th.

TABLE II.

Exp. No.	Born.	Seen in copula.	Eggs.	Larvæ.	Pupæ.	Imagines.
I....	28. 1. 12	30. 1. 12	3. 2. 12	6. 2. 12	24. 2. 12	26. 2. 12
II....	26. 2. 12	27. 2. 12	1. 3. 12	4. 3. 12	9. 3. 12	11. 3. 12
III....	15. 4. 12	16. 4. 12	21. 4. 12	23. 4. 12	28. 4. 12	30. 4. 12
IV....	24. 4. 12 15. 4. 12	*vide* No. III.	28. 4. 12	1. 5. 12	17. 5. 12	18. 5. 12
V....	30. 4. 12	2. 5. 12	6. 5. 12	8. 5. 12	17. 5. 12	19. 5 12
VI....	20. 5. 12	22. 5. 12	24. 5. 12	26. 5. 12	30. 5. 12	31. 5. 12
VII....	4. 6. 12	5. 6. 12	7. 6. 12	10. 6. 12	15. 6. 12	16. 6. 12

N B.—All the mosquitoes from Experiment No. VII. died either during their emergence from the pupae or shortly afterwards. A great number of the pupae themselves died. This was probably due to the excessive heat (Max. shade temp. 100–109° F.).

THE LENGTH OF LIFE OF *PHLEBOTOMUS* IN CAPTIVITY. A NOTE ON A METHOD OF KEEPING THE FLIES ALIVE FOR EXPERIMENTAL WORK.

BY C. M. WENYON, M.B., B.S., B.Sc.

DURING the past summer I had an opportunity, while in Malta, of conducting some experiments on the best means of keeping alive the sand-fly in captivity. It is generally admitted that these flies die very easily when kept in unnatural confinement, and for this reason it is often assumed that even in nature they are very short-lived. While in Bagdad, some four years ago, I discovered a method of keeping mosquitoes alive in the hottest weather through having observed that, during the heat of the day, mosquitoes could be found resting on the outer surface of the large porous earthenware filters which are used in every house for filtering and cooling the water. Accordingly I imitated this principle on a smaller scale, with the difference that the mosquitoes were placed inside the vessel and the water outside. I obtained small porous earthenware vessels like ordinary flower pots. These were covered with fine mosquito-netting tied round by means of tape. The mosquitoes were admitted through an opening made by pulling up the mosquito-net covering at one side. When the mosquitoes had entered, the covering was re-adjusted and the vessel was placed in a shallow dish of water. The water naturally soaked up the porous earthenware, so that there was constant moisture present on the sides of the vessel, and this was greatest, of course, nearest the water in the dish. Further, just as in the case of the porous filter, evaporation from the surface tended to keep the temperature below that of the outside air. The mosquitoes within the vessel were thus able to select a suitable spot on which to rest. The mosquito-net covering over the wide mouth admitted plenty of fresh air, so that the insects within were able to enjoy life in a cool moist atmosphere with good ventilation.

In order to feed the mosquitoes I made use of a mosquito-net (size about 7 × 7 × 7 feet) which was fixed up in a room. The vessel containing the mosquitoes was taken into the net and the mosquitoes liberated. They were then easily taken up separately in small glass tubes in which they could be fed by inverting the tube on the skin. After feeding, the mosquitoes were returned to the porous vessel. In this manner I found that mosquitoes could be kept alive longer than was the case with any other method of confinement. Another advantage is that the water in the dish protects the insects in the vessel from the ravages of ants.

This method, which I found to be so good for mosquitoes, I applied to the sand-fly, *Phlebotomus*, in Malta, with equally good results. For covering, the finest muslin had to be used, for the sand-flies would force their way through the openings in the smallest meshed mosquito-netting I could get. The flies were liberated every second, third, or fourth day into a large net, from which they were easily caught separately in small glass tubes ($2 \times \frac{3}{4}$ inch) for feeding. The sand-fly almost invariably flies on to the upper cotton top of the mosquito-net, so that there is not the least difficulty in catching it in the tube and no danger of its making its escape at the bottom of the net, where it rests on the floor. It is useful to have a small table inside the mosquito-net on which to place the vessel and tubes while one is transferring the flies. They generally feed quite readily when the glass tube is inverted over the skin and in this way, by feeding the flies individually, it is possible to observe and control the feeding act, and to be certain that every fly has actually fed, a fact which is of great importance in experimental work.

The following is a diary of a batch of seven *Phlebotomus* kept in the manner just described. This was the best result obtained :—

June 20.	Seven females and two males were captured in the Dog's Home, and confined in the jar as described after the females had fed on arm.
June 23.	One male was dead, one escaped, while all seven females fed on arm.
June 25.	Seven females fed on arm.
June 27.	Ditto. Ditto.
June 29.	Ditto. Ditto.
July 1.	One female was dead, having been crushed between gauze-covering and edge of jar. Six females fed on arm. Six males were also introduced into the jar.
July 4.	Six females fed on arm, (All males are dead.)
July 6.	Ditto. Ditto.
July 9.	One female dead—remaining five fed on arm.
July 12.	Four females fed—one refused food and was dissected.
July 16.	Four females fed.
July 18.	Three females fed—one dying and was dissected.
July 21.	Three females fed on arm.
July 24.	Ditto. Ditto.
July 27.	Ditto. Ditto.
July 30.	Two females fed—one had died.
August 2.	One fed on arm—other fly dead.
August 5.	Remaining fly fed on arm.
August 8.	Fly had died between Aug. 5th and 8th.

During the whole of this time no eggs were laid by the flies, though on dissection they were full of well-developed eggs. It is probable that a suitable medium for their egg-laying was not present. One fly was kept alive for over 46 days, and as this fly had not been raised from the egg it is impossible to state its exact age, which must have been greater than the 46 days shown by the experiment..

MISCELLANEOUS NOTES FROM THE ENTOMOLOGICAL DEPARTMENT.

These include :—

(1) *Anopheles umbrosus*, Theobald, as a Natural Carrier of Malaria in North Borneo ; reported by Dr R. Roper.

(2) An Earwig (*Dicrana frontalis*, Kirby) as a "Stinging" Insect ; reported by Dr. Yale Massey.

(3) A case of Intestinal Myiasis (*Sarcophaga* sp.) in a Chinawoman in Hongkong ; reported by Dr. G. E. Aubrey.

(4) A supposed case of Scarabs in the Nasal Passages ; reported by Dr. R. E. Duke-Brockman.

1. *ANOPHELES UMBROSUS*, THEOBALD, AS A NATURAL CARRIER IN NORTH BORNEO.

DR. R. ROPER has sent to the School specimens of this species, which, he states, is common in the hospital at Membakut in North Borneo. He was particularly anxious to have the species correctly identified, as in one specimen he had found the salivary glands infected with sporozoites. This is an interesting fact since, although Dr. Malcolm Watson includes *Anopheles umbrosus* among the natural carriers in the Malay States, Dr. A. T. Stanton, in several experiments at the Kuala Lumpur Laboratory was unable to infect the species with the parasites of subtertian malaria. These inconsistent observations and experiments once more impress the necessity of continued research, in divers places and at different seasons, in order to establish the pathogenic relations of any particular species of *Anopheles*.

It may be mentioned, incidentally, on the authority of Dr. Roper, that although *Anopheles kochi*, Dönitz, is very common at Membakut, and is frequently found there in houses, and is suspect on epidemological grounds, none of the specimens dissected by him, so far, has shown any actual evidence of infection.

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2. AN EARWIG (*DICRANA FRONTALIS*, KIRBY) AS A "STINGING" INSECT.

DR. YALE MASSEY has brought to the School, from the Congo, an Earwig which he caught, *in flagrantissimo crimine*, "biting or stinging" him on the neck.

"The sensation was a burning and itching, which lasted a couple of hours, and the part was sensitive to the touch for many hours after. There was no swelling and no mark, other than a diffuse redness about the size of a florin. The little beast is well known among the natives."

Mr. Meade-Waldo of the British Museum, in identifying the insect as *Dicrana frontalis*, Kirby, refers to the fact that an American Earwig (*Doru lineare*, Esch.) has been accused of inflicting a bite that causes considerable urtication.

It is not always, in cases of this kind, that the evidence is furnished by a witness of such experience and competence as Dr. Yale Massey.

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3. A CASE OF INTESTINAL MYIASIS (*SARCOPHAGA* SP.) IN A CHINAWOMAN IN HONGKONG.

BY DR. G. E. AUBREY.

"THE two flies forwarded with this I hatched from a case of Myiasis: they belong, I think, to the *Sarcophagidæ*. The history of the case is as follows: Woman, aged 43, Chinese, had abdominal pain, anæmia, and a very foul mouth for two years, with alternate attacks of constipation and diarrhœa. Fæces taken direct from the rectum contained an enormous number of *fly-eggs, hatched and unhatched*, also larvæ. Some 'full' eggs kept under observation hatched in a few hours. The larva has on each segment 7 or 8 rows of fine hair-like projections: the larval stage lasted 17 days: the pupal stage lasted 10 days. [The fæces also contained *Ascaris lumbricoides* eggs, *Trichocephalus dispar*, and *Clonorchis sinensis*.] The fly-eggs were very resistant to treatment, a few being found 16 days after the patient had been treated with santonin, resorcin and salol, chloroform and oil of eucalyptus: a few days after administration of the last they disappeared, and there was considerable improvement in the patient's condition."

The flies are a species of *Sarcophaga* which seems to be undescribed.

4. A "case" of Scarabs in the Nasal Passages.

By Dr. R. E. DRAKE-BROCKMAN.

One frequently meets with foreign bodies in the nasal passages among natives in the Tropics, but I am unaware that a scarab has ever before been reported.

According to the Somalis, although the entrance of a beetle into the nose during sleep is of comparatively rare occurrence, most of them are acquainted with cases both in men and women.

The patient, a woman, aged about sixty years, states that one year ago, while asleep on the ground in the jungle, a scarab entered her nose and all her efforts to get it out failed. Two months later she came to Bulhar, where an old woman who had a great reputation for getting rid of scarabs from the nose, lived. She consulted the old woman, and after partaking of a meal of meat with which a certain root had been boiled, she vomited up forty-one small scarabs. The following was the treatment prescribed:—

Some mutton was first boiled, and while still boiling the root of a certain plant, only known to the old woman, was pounded up and added to the meat in the pot and the whole stirred up. As soon as the meat was cooked it was eaten but the soup had to be allowed to cool down before being swallowed. As soon as the cold soup entered the stomach, the patient vomited bringing up the scarabs.

According to the patient, the smell of the meat which was first eaten attracted the scarabs down from the nose into the stomach, while the cold soup swallowed some little time after, caused her to vomit up the contents of the stomach together with the scarabs.

Each time the patient comes down to the coast she partakes of this treatment and brings up a number of scarabs. The scarabs are always alive when vomited.

Doubtful of the truth of the above statements I placed the afflicted woman under chloroform in the hope of destroying the remaining scarabs in the nasal passages, but on recovery from the effects of the anæsthetic the patient was unable, although she vomited several times, to produce any scarabs.

Dr. Drake-Brockman, in a letter accompanying a number of specimens of scarabs "said to have been brought up by the patient," states that the Somalis get annoyed with him when he doubts the story, and affirm its truth strenuously. As Dr. Drake-Brockman himself had observed, the scarabs in question belong to two distinct species!

III.—HELMINTHOLOGY.

SEVEN HELMINTHOLOGICAL NOTES.

By R. T. LEIPER, D.Sc., M.B., Ch.B., F.Z.S.

(a) THE OCCURRENCE OF GUINEAWORM IN NYASALAND.

IN a paper on "Guineaworm in domesticated animals," published in the 'Journal of Tropical Medicine' for 1st March, 1910, I drew attention to the curious distribution of Dracontiasis in Africa and placed on record the first instance of an infection acquired apparently south of the Equator. The specimen came from a leopard shot by Mr. C. Grey at Broken Hill, N.W. Rhodesia, and had been forwarded by him to the London School of Tropical Medicine.

Recently Dr. Drummond has presented to the School two guineaworms extracted by him from patients under his care in Nyasaland. Both came from the same village and had, in his opinion, undoubtedly acquired their infections there.

These two records seem of some significance, and the matter will be, doubtless, the subject of local inquiry in view of the large economic loss which the spread of Dracontiasis always entails.

Dr. Drummond informs me that there is a military station about sixty miles from the infected village and that Indian troops are posted there. It is not at all unlikely that cases of Dracontiasis appear sporadically amongst these troops, the infection having been contracted in India, but at present, without further information, it is impossible to say whether the endemic cases arose primarily in this way or if the infection was from the West or from the Sudan, where it is so common.

(b) NATIVE SUPERSTITIONS REGARDING GORDIAN WORMS.

SOME time ago I published a note to show that a large Gordius worm in West Africa was associated in the native mind with the causal agent of Dracontiasis. A short time ago Dr. Stannus forwarded to me a similar worm, and at the same time contributes the following interesting notes :—

The worm is known to the natives by the name Kasipa and is found in pools in streams, after the heavy rains have stopped (April–May). It is said by natives to be the cause of a disease of the same name, Kasipa.

(1) It is said to enter the body and cause the disease.

(2) Natives will not handle it for fear of getting the disease.

(3) Medicine is made of the dead worm and put in the pathway, while the name of the person whom it is wished to attack is uttered ; it is then said to have selective action and cause the disease in the person named when he walks over it.

(4) Scott, in his "Mang'anja" Dictionary, says that the remains of the medicine which have been used to cure the disease are buried at the foot of a tree that the disease may be transferred to the tree : anyone touching that tree gets the disease.

The disease Kasipa is variously described as a swelling of the legs, ulceration of the legs, wasting of the legs, general bodily illness with neuritic or rheumatic pains.

The cases seen by Dr. Stannus have turned out to be syphilitic and spirochætal ulcers, Malaria, and possibly Beri-Beri. One native described multiple transient swellings, apparently resembling Calabar swellings, as a symptom. There is of course no evidence to connect the worm with the disease, but it is of interest to find that these Gordian Worms have attracted notice and have established themselves in the folk-lore of various parts of Africa as well as at home. In Britain these worms form the basis of the childish fable that hairs from a horse's tail, if left in a pool of a sheltered stream, will turn into living worms in a week or so!

(c) THE OCCURRENCE OF *HETEROPHYES HETEROPHYES* IN CASES FROM THE FAR EAST.

DURING the Summer Session I received, through the Pathological Department of the School, material from two cases under treatment in the Albert Dock Hospital. In one case, that of a Chinaman, a fragment of a minute fluke had been noticed by one of the students. From an examination of this I concluded that the worm was a species of *Heterophyes*, and as numerous eggs were present in the faeces, the bulk of the stool was carefully inspected in thin layers by means of a hand lens. Two hundred specimens of *Heterophyes* were recovered. A short time afterwards a Japanese officer was admitted suffering from dysenteric symptoms. From a sample of faeces sent for examination I diagnosed the eggs of *Heterophyes*. After treatment with Magnesium sulphate and Thymol sixty worms were obtained.

It is interesting that a number of the worms were found in the stool following the preliminary course of Salts.

The worms correspond in every respect with the *Heterophyes heterophyes* which has been recorded several times from Egypt. The eggs are described by Looss as a light brown. In the second case they were of a deep brown colour, and made the worms easily visible as little black flecks lying upon the surface of the fæces.

(d) FLUKES IN THE DOG IN ASIA.

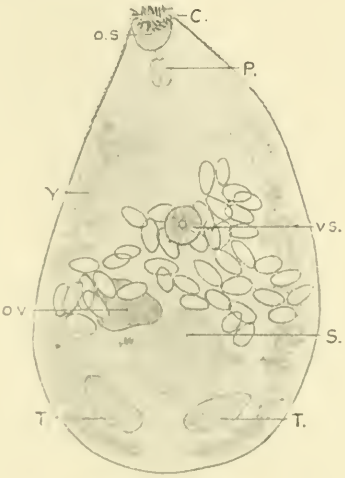
MAINLY through the keenness of a former student, Dr. O'Connor, I have recently been able to examine material containing three species of flukes from dogs in Formosa. The specimens were obtained in the first place by Dr. S. Yokogawa. The trematodes recorded hitherto from the dog in Asia are: *Schistosoma japonicum*, *Paragonimus westermanii*, *Clonorchis endemicus*, *Opisthorchis noverca*, and *O. caninus*.

In a recent paper of "Observations on certain Helminths in Man," I have expressed the view that the two last-named parasites belong to one and the same species. We have, then, four well recognised parasites. To these no less than three have now to be added, viz.:—*Heterophyes heterophyes*, *Yokogawa yokogawa*, and a new species, *Centrocestus cuspidatus*, var. *caninus*, mihi.

It is a striking fact that, with the exception of the last, all these flukes are parasitic also in man. The occurrence of *Heterophyes* in man in the far East is recorded in the preceding note. *Yokogawa yokogawa* is stated by

Dr. S. Yokogawa to be sporadic in man and dog over Korea, Japan, and Formosa. The species of *Centrocestus* is represented at present by a single example in a slide of fæces from the dog prepared by Yokogawa. It seems probable that it will be found as a parasite of man if careful search is made.

The genus *Centrocestus* contains but one species, *cuspidatus*. This fluke was discovered by Looss, in Egypt, in the intestine of the Kite, *Milvus parasiticus*, and first described by him as *Distomum cuspidatum* in 1893, in his "Recherches sur la Faune parasitaire de l'Egypte." In one respect the specimen from the dog differs from that described and illustrated by Looss: whereas, in *D. cuspidatus*, there are 36 spines around the mouth, there are in my specimen only 28. The worm, however, is not perfectly preserved, and some of the spines may have been lost. There is a discrepancy in the relative size of the testes, but this also may be due to maturity. A similar variation can be made out in specimens of *Heterophyes*, *Yokogawa*, and "*Paropisthorchis*." We are then faced with this difficulty:—Does the same species inhabit the Kite in Egypt and the dog in Formosa? Looss has already had this problem in another species, and has upheld the view that although his forms were apparently morphologically identical, they must be regarded as physiologically distinct species on account of the difference in habitat and geographical distribution.



Centrocestus cuspidatus, var. *caninus*. From the Dog in Formosa.

C. Circlet of spines.
P. Pharynx.
O.S. Oral sucker.
O.V. Ovary.
S. Seminal vesicle.
T. Testes.
Y. Yolk-glands.

I follow his ruling in this case to the extent that I name the Formosan *Centrocestus* a distinct variety, viz *Centrocestus cuspidatus*, var. *caninus*. When further material is available, and some headway has been made with the study of the helminth fauna of the birds of Formosa, the status of *caninus* as a variety or species can be decided.

The accompanying text-figure was drawn from a direct projection of the specimen.

(*e*) OCCURRENCE OF *HYMENOLEPIS DIMINUTA* IN A CHILD IN GRENADA.

DR. R. P. COCKIN has presented to the School a specimen of *Hymenolepis diminuta* obtained from a child, aged 5 years, after treatment with Extractum Filicis liquidum. The child had been under observation for about nine months, and suffered from pain in the epigastrium. The blood showed a slight eosinophilia, while in the fæces *Trichocephalus* eggs and those of a cestode had been noted. The cestode egg did not correspond to any figured in the textbooks, but some were undoubtedly those of *H. diminuta.* Compared with *Hymenolepis nana*, this species is rare in man, but its distribution is a wide one, as might be expected from the fact that rats and mice are the normal hosts.

(*f*) OCCURRENCE OF *PARAGONIMUS WESTERMANII* IN INDIA.

DR. H. G. WATERS, who has just returned to England on leave, informs me that an undoubted case of Paragonimus in a native recently came under his notice in Central India. This record is deserving of note in view of the possibility that the Paragonimus may have been the same as that described by Cobbold as *Distomum compactum* in the Mongoose (*Viverra mungos*). Paragonimus appears to have a wide distribution in Asia and to invade various hosts, *e.g.*, Tiger, Cat, Dog, Pig, Man.

Should, as I have long suspected, the *Distomum compactum*, of Cobbold, be the same species as *Paragonimus westermanii*, the former nomenclature has priority, and the name would thus become *Paragonimus compactus.*

(*g*) NOTE ON THE SPICULES OF *NECATOR AMERICANUS.*

IN a note in the 'Journal of the American Medical Association.' Stitt has drawn attention to the fact that the two spicules in *Necator americanus* are constantly fused. This I have recently verified, and find that the spicules differ in several respects from those of *Ancylostoma duodenale.* They are provided with wide lateral expansions which are transversely striated, giving each spicule the appearance of a triangular file. These expansions gradually disappear, and the spicules when united show a delicate smooth ledge without striæ, but slightly granular. This continues to the tip and forms the triangular piece. The rod-like portions of the spicules support this cuticular blade, one spicule being slightly curled to give support to the proximal angle of the barb, the other continuing for some distance towards the extreme point.

IV.—PATHOLOGY.

H. B. NEWHAM, M.R.C.S., D.P.H., &c.

The Superficial Lymph-nodes as a Source of Leishmania for the Diagnosis of Kala-azar; with some observations on Kala-azar in China.

By S. COCHRAN, M.D., China.

For many years the devastating epidemics of fever occurring in certain parts of India associated with marked splenic enlargement were a puzzling problem which was cleared up about ten years ago by the discovery of the parasite causing the condition, the Leishman-Donovan body. This discovery further made possible the demonstration of the fact that the disease had a much wider prevalence than had been supposed, and is endemic along both shores of the Mediterranean and in the Sudan. Some years ago scattered cases were reported from China, but it is only recently that we have begun to realise how widely extended is the Chinese endemic area, and how large must be the aggregate number of cases.

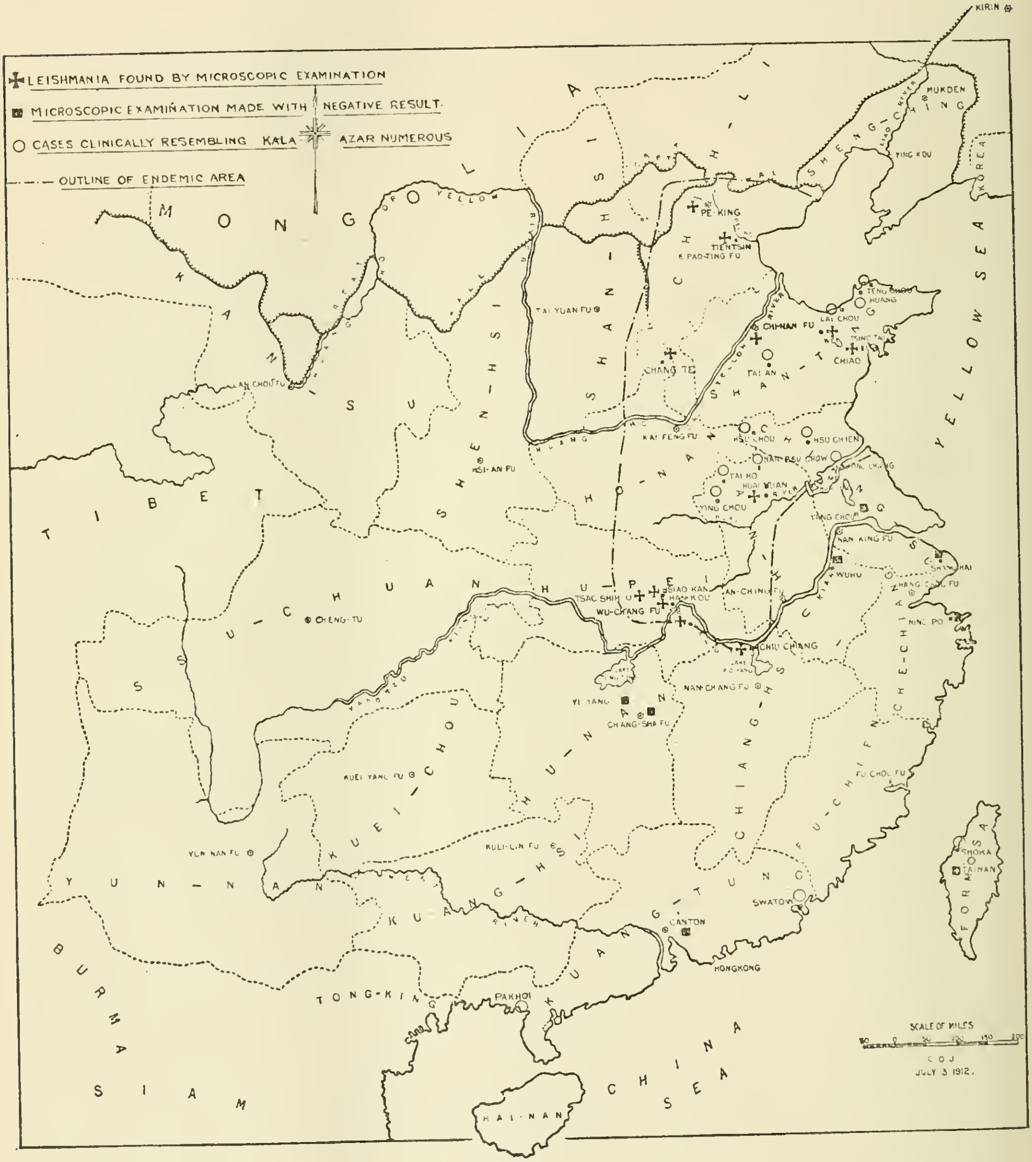
Distribution of Kala-azar in China and Korea.

Last year I made an attempt to obtain accurate information of the distribution of the disease. A circular-letter was sent to all physicians, whose names could be obtained, practising in China and Korea. The province of Chihli was omitted, as I had information that a similar canvass was already contemplated for that province by a resident there.

Some 70 answers have been received; of these, the following men report having found the parasite:—*Hupeh*: Aird, of Hankow, Fowler, of Hsiaokan, and Thomson, of Hankow, by spleen puncture; *Honan*: Leslie, of Changte, by spleen and liver punctures; *Shantung*: Schultz, of Tsinan, by smears from excised lymph-nodes, and smears from an excised spleen; and Roys, of Weihsien, by spleen puncture and excised lymph-node. I have verbal communications also from Lambert, of Kiukiang, Somerville, of Wuchang, and Wills, of Tsaoshih, *Hupeh*, telling of finding the parasite in spleen punctures. To these may be added Aspland, of Peking (1), and a case in Tientsin, also mentioned by Aspland (2). Material from cases resembling Kala-azar clinically have been examined microscopically with a negative result in the following places:—Ningpo (two cases of spleen puncture and one of liver puncture); Tainan, *Formosa* ("not a few cases" of splenic and hepatic puncture): Yiyang, *Hunan* (circulating blood); Changsha (method not stated); Changteh, *Hunan* (by gland smears); Shanghai (spleen puncture and circulating blood): Yangchow (lymph-nodes); Canton (spleen and circulating blood); *Hainan* (liver, spleen and blood); Wuhu, *Anhwei* (numerous liver and spleen punctures).

Cases resembling Kala-azar are not seen in the following localities:—*Anhwei*: Luchowfu; *Fukien*: Kutien, Kienking; *Hunan*: Hengchow, Shengchow; *Kwangtung*: Hongkong, Kongmoon; *Szechwan*: Tungchwanfu, Chungking, Mienchuhsien, Yachow; *Manchuria*: Chaoyanghsien, Kwangchengtze, Liaoyang, Kirin, Tiehliang, Yungling, Antung; *Korea*: Haiju, Chemulpo, Seoul, Kunsan.

Cases clinically like Kala-azar are seen in moderate numbers in the following places:—*Chekiang*: Shaowu, Wenchow; *Fukien*: Fuchow; *Hunan*: Changteh; *Kiangsi*: Jaochow, Suchow; *Kwangtung*: Takhing, Yungkong, Dosing; *Shensi*: Sianfu; *Szechwan*: Paoning, Tzeliutsing; *Shantung*: Pangchwang, Pinying, Lintsingchow.



There have come reports that cases clinically resembling Kala-azar are seen in large numbers in the following places:—*Kiangsu* : Sutsien, Tsingkiangpu, Hsuchowfu ; *Shantung* : Taian, Tengchow, Laichowfu, Hwanghsien ; *Formosa* : Shoka ; *Kwangtung* : Pakhoi, Swatow.

It will be noticed from the above, by comparing with the map, that all the positive examinations have been reported from north of the Yangtze, with the exception of Wuchang and Kiukiang, which lie on the river. All the negative examinations, on the other hand, lie to the south of the Yangtze, with the exception of Yangchow, only a few miles from the river. Furthermore, cases clinically diagnosed as Kala-azar are seen in large numbers at several points in Kiangsu and Shantung, connecting the points in *Anhwei*, *Shantung*, and *Chihli*, where a positive diagnosis has been made. I have personal knowledge that there are large numbers of cases giving typical Kala-azar symptoms in Nanhsuchow, Ingchowfu, and Taihohsien, in *Anhwei*. On the other hand, the numerous cases reported from Swatow, Pakhoi, and Shoka (Formosa), if interpreted in the light of the negative examinations reported in similar cases in their neighbourhoods, would seem not to be Kala-azar. In the light, then, of the somewhat meagre information now at hand, the endemic area of Kala-azar in China will be somewhat as indicated by the outline on the map.

Incidence of Kala-azar in Hwaiyuan.

Anyone in charge of a general hospital service in this part of China must be struck at once by the large number of cases with splenomegaly applying for treatment. Of course some of them are undoubtedly due to other causes; the first case, for example, that we took into the hospital ward to examine carefully for possible Leishmania, turned out to be splenomedullary Leukæmia. Making due allowance, however, for such exceptions, the majority of cases of large spleen applying for treatment are with little doubt due to Leishmania. It is, of course, impossible to make any estimate of the number of cases in proportion to the general population. A little idea of it can, however, be obtained from the fact that in two years, from June 1st, 1910, to the corresponding date 1912, out of a total of 8058 different patients applying for treatment to our out-patient department, 83 are recorded under the diagnosis of "Kala-azar," or "Splenomegaly" ; a little over one per cent.

Age.

The average age of these patients on applying for treatment was 17·4 years. Divided into decades they are classified as follows :—

		per cent.
1–10 years	27	32·6
10–20 „	24	29
21–30 „	18	21·6
31–40 „	11	13·2
41–50 „	3	3·6
	83	100

51·6 per cent. therefore are under 20 years of age.

The oldest patient was 50 years, and the youngest, 2 years. Of these 83 patients a positive diagnosis was made in 20 cases by the finding of Leishmania, and in 16 of these the average age on admission to the hospital was 16·8 years, the youngest case being four years old, and the oldest 35.

Experience in other places in China where the positive diagnosis has been made is as folllows :—

Hankow. Aird and Thomson's cases were adults.
Ksiaokan. One patient of 22, and one of 57.
Weihsien. Mostly children, though cases in adults not uncommon.
Tsinan. Both children and adults.
Changte, *Honan.* Leslie reports that his cases are children.
Peking. Aspland's cases are exclusively among children.

It is very evident, therefore, that the disease is not Kala-azar Infantum, so-called, unless in the northern part of the area. As it so certainly includes adults in considerable proportions, in a large part of the area, and as the area is apparently continuous, one wonders if perhaps adult cases may not in time be observed throughout. In fact, there hardly seems sufficient positive evidence to warrant the dividing the disease anywhere into two separate entities, Kala-azar and Kala-azar Infantum.

Sex.

Much the larger number of our patients were male, only four of the 83 being female. This is not an accurate indication of the true division between sexes, as more men than women apply to us for treatment for most diseases. Moreover, for a small part of the time covered by the statistics, female cases were being treated at a different dispensary, and the statistics were drawn from the male service alone for this time. The fact remains that there is an apparent large preponderance of male cases.

Duration.

In fourteen cases positively diagnosed, in which we have record of the duration, it averaged 16·8 months from onset to application for treatment. The shortest duration was four months, and the longest 32. Of 28 cases of which we have record, which include some only diagnosed clinically, the average duration was 12·8 months, the shortest being four months, and the longest five or six years. With the exception of two, no case had lasted three years. Our only fatal case had lasted twelve months.

Time of Onset.

Accurate histories are rather hard to obtain, as many of the patients are hazy about when the trouble began. Out of 36 cases whose report we have, the larger number began in the spring or the early summer. By months the record is as follows :—January, 2 ; February, 2 : March, 4 ; April, 1 ; May, 6 ; June, 6 ; July, 5 ; August, 1 ; September, 3 ; October, 2 ; November, 4. Nearly half the cases (seventeen), therefore, began in May, June, and July.

Family and Local Incidence.

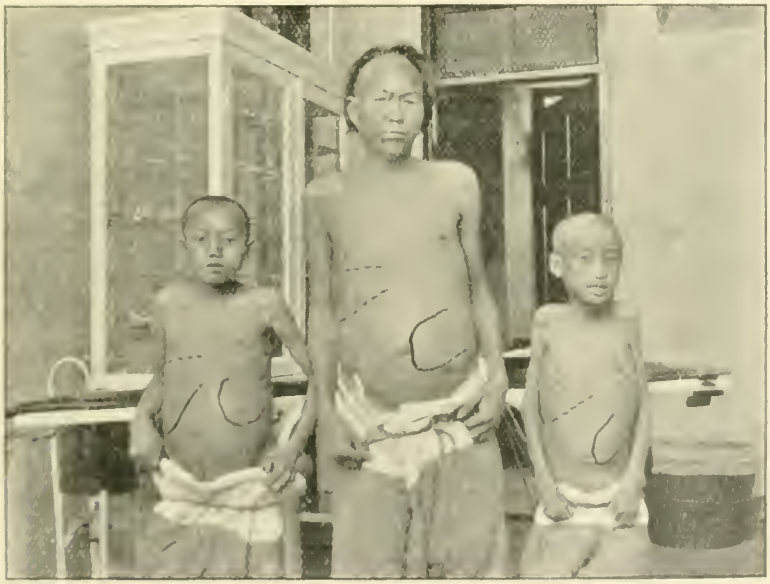
Of twelve cases positively diagnosed, four give a history of other cases in the same family, five know of other cases in the same village, and three know of no such cases. Of sixteen other patients, in whom the diagnosis is merely clinical, nine knew of no other cases in the neighbourhood, five had cases in their own family, and two knew of cases in the village. While not striking, these figures would indicate the same tendency to grouping in families that has been observed elsewhere.

Transmission.

There is little to be gleaned from our cases on this point. There are enormous numbers of the commoner parasites found everywhere in this district, namely, fleas, lice, bed-bugs, and mosquitoes. Lice are active all the year round. The cold weather in this part of China lasts until March, and about the first of April bed-bugs begin to be a nuisance in the native houses and inns. Fleas, also, are not annoying in winter, nor are mosquitoes. The increased number of infections in the months of May, June, and July, may have some reference to this spring awaking of the above pests. Attempt has been unsuccessful to obtain dogs for examination from houses where Kala-azar exists.

Symptoms.

Onset. Fever.—The first symptom is usually fever and is thought by the patient to be malaria. It is often accompanied by chills. It is, however, irregular in character and rarely, if ever, do the patients fail to remember that the periodicity of malaria is lacking. Out of fourteen cases diagnosed positively, twelve began with fever, one first noticed the enlarged spleen, and in one case the two were noticed at once.



Illustrating the splenic and hepatic enlargements in three cases.

Spleen.—Usually the spleen is noticed to be enlarged at an interval of from ten days to a month after the beginning of the fever, though in one case it was not noticed for nine months, and another six months, after the fever began. When coming to the dispensary, the splenic tumour together with the cachexia and general ill-health are the troubles for which they usually desire treatment, though occasionally advanced cases come because of a cancrum oris, or some other complication. Thus far we have failed to diagnose any cases in an early stage before the spleen's enlargement.

Diarrhœa.—Though some of the cases have diarrhœa, it has not been a particularly characteristic symptom. In only one case was it severe and persistent. Its presence is recorded as part of the history of seven cases at one time or another, but in most of them it was only slight and occasional, and in eight other cases it was lacking; diarrhœa is so common in this neighbourhood that probably an equally high percentage would be found among the rest of the population.

Cancrum Oris.—This is a not uncommon termination of the disease in children, but we have not seen it in adults. Kala-azar is the most common cause of cancrum oris with us. There is probably nothing specific about it as it also occurs following other diseases causing diminished vitality, such as measles. It usually begins with an ulcer on the inside of the cheek, followed by a black spot, and not, as has been observed elsewhere, around the incisor teeth. It was the cause of death in the only case that died in the hospital, and though we have seen a good many cases in the dispensary we have known of none to recover, though of course most of them have not been kept under observation.

Hæmorrhagic Symptoms.—Out of fifteen cases, ten gave history of having had epistaxis, some of these being frequent, and of these two had had bleeding from the gums; another case without epistaxis had had bleeding from the gums, and four gave no history of hæmorrhagic symptoms. There was evident, however, in taking out the lymph-nodes for examination, a tendency to hæmorrhage with slight evidence of its spontaneous checking. The wounds, however, made by this slight operation healed in the usual manner with the exception of three cases, in one of which the wound was kept open by a malingerer.

Deafness.—Partial deafness has been observed in several cases.

Examination.

Appearance.—The appearance of the patients is very characteristic. The most marked thing about it is the general expression of malaise and distress, and entire lack of vivacity. The colour is pale, with often a slight subicteric tinge. With us there is no trace of the darkening of the skin that gave the disease its name in India. As elsewhere, there is emaciation and enlargement of the abdomen.

Spleen.—The enlargement is much more marked, on the average, than from malaria, or any of the more usual causes of splenomegaly which we see, such as typhoid, typhus, or relapsing fever. In nine of the cases positively diagnosed, it is noted that the spleen tumour is as follows:—reaches navel, 4; almost reaches navel, 2; half-way to novel, 2; less than half-way to navel, 1. Of cases clinically diagnosed we note:—reaching navel, 2; almost to navel, 13; half-way, 7; less than half-way, 4. Six cases were measured, and the spleen was of an average diameter of 11 cm., the smallest being 8 cm.

and the largest 20 cm. When under observation there is more or less variation in its size, increasing with exacerbations, and diminishing with remissions.

Liver.—In twelve cases positively diagnosed the liver was palpable in eight, and not made out to be enlarged in four.

Lymphatic Glands.—The inguinal lymphatics have been more or less enlarged in every case examined. Usually the enlargement has not been noticeable and perhaps not much if any greater than is often found in perfectly healthy individuals. There has been much variation about the other glands. Out of ten cases where note was made, the inguinal were enlarged in all, the axillary in six, the epitrochlear in four, and the anterior and posterior cervical in one each. Occasionally the enlargement is considerable, to the size of a fair-sized bean. One or more have been excised in 21 cases. They are firm, show no tendency to break down, and when cut exude a pale serum. Staining shows this to contain mainly mononuclear cells of various sorts. Unless contaminated with blood from the outside surface of the gland, there are few, if any, erythrocytes in the smears.

Fever.—In the stage in which we have seen the patients there is a low, irregular fever, with occasional exacerbations of higher temperature.

Blood Examination.—The following is a synopsis of the blood examination, on admission, of a number of cases in which a positive diagnosis was made.

The differential count in cases with so few leucocytes is very time-consuming, and we accordingly only counted 100 * leucocytes in each case, so there is doubtless a small margin of error. That it is not very large, however, is indicated by the close way in which the results in the different cases agree. There is only a variation of from 43 to 55 per hundred in the polynuclears.

Diagnosis.

Clinically the diagnosis is easy in the cases that come to us, as the symptom complex in this stage is very characteristic. If to the history and the physical examination be added the result of an examination of the blood, mistakes will seldom be made, as the blood picture is almost as characteristic as are the other symptoms. As Rogers points out, there is almost invariably a marked leucopenia, both absolute as regards the number of white cells per c.mm., and relative as regards the ratio of white cells to red (3). A positive diagnosis is, however, impossible without the demonstration of the parasite, and it is often desirable that such a diagnosis should be made. For example, reports of successful treatment will lack most of their validity failing the positive knowledge that the cases treated are really Leishmaniasis. Again, as Rogers suggests, a positive diagnosis may be of importance in getting patients to submit to a long and distasteful course of quinine medication (4).

It might also be necessary before advising quarantine measures involving financial outlay or destruction of property. Unfortunately, there is no such ready and reliable source of the parasite as there is in relapsing fever and malaria. A number of methods have been tried and three of them are in general use, but to them all there are important objections.

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* Rogers only advises counting 125 in these cases. 'Fevers of Tropics,' p. 21.

TABLE A.

Case.	Red Cells.	White Cells.	Ratio, White to Red.	Large Mononuclears.	Lymphocytes.	Polymorpho-nuclears.	Eosinophiles.
				per cent.	per cent.	per cent.	per cent.
I.	....	4040	....	31	16·5	43·5	4
II.	....	906					
VII.	1,560,000	1560	1/1000	26	15	51	7 1 mast cell.
VIII.	2,780,000	2333	1/1190	38	17	43	1
IX.	1,380,000	1500	1/920	34	13	51	2
X.	2,716,000	2240	1/1210	33	21	43	1 2 mast cells.
XI.	1,382,000	1100	1/1250	49	6	45	0 1 nucleated red cell.
XII.	....	1300					
XIII.	....	5400	....	15	30	55	
XVII.	3,340,000	4950	1/670	44	8	45	3
XVIII.	2,976,000	1352	1/2200	23	30	47	0
XX.	....	1660					
Average	2,304,860	2360	1/1200	32·5	17·5	47·5	2·1

In cases III., IV., V., VI., XIV., XV., XVI., and XIX. no examination of the blood was made.

1. *Examination of Smears from the Circulating Blood.*—Though Donovan (5) and Marshall have found the parasite by this means in a high percentage of cases, the general experience of other observers is that the method is uncertain and excessively laborious. Most other practitioners have failed to find it in anything like as large a percentage of the cases. The French and Italian observers have had little success with the method (6), and Rogers finds it necessary sometimes to resort to visceral puncture in spite of the caution he feels about doing so (7).

The amount of time which must be spent in searching for them may be judged by Marshall's statement that there did not average more than one parasite to a slide, and that "in the majority of positive cases the examination of three slides is usually sufficient for diagnostic purposes" (8). The examination of three slides on the average would seem to take an almost prohibitive amount of time and rule out the method as a routine procedure.

In our own cases we made a trial of blood-smears in three of our early cases but without success, and had to adopt other means for demonstrating that the local endemic of splenomegaly was due to Leishmaniasis.

2. *Splenic Puncture.*—This is at present the most used procedure. Its safety is questioned by some, and all advise that it be used with caution because of the many recorded fatal cases. To quote Rogers, "In Kala-azar so many fatalities have followed that much caution is necessary in using it in that condition" (9). Manson (10) and Bassett-Smith (11) point out the danger in using it, and Donovan has abandoned it (12). Out of a dozen men in China, from whom I have word that they have used this method in diagnosing suspected Kala-azar, two have reported fatal results, in a total of three cases.

Further, diagnosis by this method is not very certain and often laborious. Bousfield says, in reporting eight cases positively diagnosed:—"In the first case, it was not till the third splenic puncture that parasites were seen, and in several others the results from the first and second punctures were negative. Often only one film out of four or five would present parasites, and even then in very small numbers. The average time spent for microscopic diagnosis has certainly worked out at nearly four hours for each case; in only three cases were parasites found in the first films examined" (13). Again he says, in reporting a series of 39 cases, "undoubtedly Kala-azar from a clinical standpoint," that only 19 showed parasites in their splenic blood, *i.e.* in about 48·5 per cent. of the clinical cases the parasite was demonstrated. "A positive spleen puncture settles the diagnosis but a negative still leaves one in doubt" (14). Nicolle reports, "En effet, il existe, en Tunisie tout le moins, des anemies spleniques infantiles de cause inconnue encore et que nous ne savons de distinguer de la maladie qui nous occupé que par le seul résultat négatif de la recherche du parasite" (15). Is it quite likely that a more reliable method of diagnosis than has hitherto been at our disposal would show many at least of these cases to be Leishmaniasis? In China the conditions are much the same, I believe. Somerville, for example, said in a verbal communication that the search of smears from spleen punctures is tedious, sometimes taking hours. My personal experience is limited to one case in which one splenic puncture proved negative because of excessive blood withdrawn, and the second only showed Leishmania after about two hours' search. Yet in this same case a lymph-node was removed two days later, and Leishmania were found in profusion in almost every field. With such an expenditure of time the method is practically only possible in the main to those who are engaged in research work, and it would be highly desirable to find some shorter means for routine examinations, to bring them within the reach of ordinary hospital services. With our own hospital, for example, searches of an average of four hours, or even approaching it, are out of the question.

3. *Liver Puncture.*—This method is generally considered to be safer than splenic puncture; but yields the parasite even less quickly and certainly than the latter. I have tried it in one case that was clinically Kala-azar but with a negative result.

4. *Blisters.*—I have tried croton-oil pustules and Spanish-fly blisters in two or three cases with negative results, and the literature shows that others have had only occasional successful results (16).

5. *Lumbar Puncture.*—Lumbar puncture (17) and puncture of the tibia (18) have been used by La Cava and Pianese but are hardly likely to come into general use, as they are about as formidable as visceral puncture.

6. *Lymph Nodes.*—I was led to attempt to find Leishmania in the superficial lymphatics by the fact that they yield trypanosomes for the diagnosis of Sleeping Sickness. In the first case a posterior cervical node was taken out under local anæsthesia, and I was astonished at the enormous numbers of the parasite found, when but two days before I had had such scanty result from a spleen puncture in the same case. I now report twenty cases in which this method has been used with nineteen positive results. Not included in this series are two cases in which gland-smears were examined with negative results, but which were not clinically straight Kala-azar. In one of these the spleen was not enlarged, but the patient was cachectic and had a white cell count of 5600. He was examined on the chance of finding a case in the period previous to the splenic enlargement. The other was a case of relapsing fever with a large spleen which had lasted for a year, but in this case also the symptoms of Kala-azar were not clear.

Out of these twenty cases 95 per cent. have given positive results in either the smears or the puncture.

The average time spent at the microscope before finding the first parasite in each case was 26 minutes. Excluding three cases in which the search was tedious, sixteen of the twenty cases showed the parasite in an average of 8·4 minutes.

It is quite possible that further search would have been successful in case No. XV., as the hunt was abandoned an hour earlier than the average time reported in one series of cases (14).

Many of the cases showed forms that were very probably Leishmania but in a degenerating condition. The negative case No. XV., for example, had doubtful forms like some of those depicted in Plate VIII. of the 4th Khartoum Report. No case was considered to have had a positive diagnosis made without the finding of a body answering to the following criteria,—a faintly staining body with distinct outline, a large nucleus and a smaller blepharoplast (the latter staining more intensely, and both of the characteristic purple colour). In the cases in a general way in which the search was brief, my impression is that the parasites were smaller and of the characteristic torpedo shape with nuclei very sharp; the slides contained few, if any, of the larger less sharply staining forms. In the cases in which the search was more tedious, these and the degenerating forms were found; one of the longest searches was in the case with the noma—a type of case that is said to spontaneously recover occasionally; the negative search was in the case of a patient that was doing very well, being a soldier on active service ; the leucocyte count averaged considerably higher in the cases that showed few parasites. These facts would lead to the conclusion that when the patient shows the tendency to recover there is a marked diminution of

TABLE B.

Case.	Source.	Time of search.		
		Smears.		Puncture.
I.	Post-cervical.	Every field.	......	Negative.
II.	Post-cervical.	5 min.	......	Not successful. No fluid drawn.
III.	......	25 „	......	Not attempted.
IV.	......	5 „	......	„ „
V.	......	5 „	Very abundant.	„ „
VI.	Post-cervical.	2 „	......	„ „
VII.	Post-cervical.	2 „	......	Positive, 10 min.
VIII.	......	½ „	......	Negative.
IX.	Inguinal.	20 „	......	„
X.	Inguinal.	4 „	Scanty.	Not attempted.
XI.	Inguinal.	8 „	Scanty.	Positive, 12 min.
XII.	Inguinal.	2 hrs.	Negative.	Positive, 40 min.
	(This case showed parasites in a gland-smear some weeks later.)			
XIII.	Inguinal.	3 min.	......	Puncture unsatisfactory.
XIV.	Inguinal.	3 „	......	Not made.
XV.	Inguinal.	3 hrs.	Negative.	„ „
	(Further examination would have been made in this case but for his refusing to stay in the hospital.)			
XVI.	Inguinal.	135 min.	......	Unsatisfactory.
XVII.	Inguinal.	2 hrs.	One degenerating parasite.	Positive, 40 min. Typical parasite.
XVIII.	Inguinal.	65 min.	......	Negative. One hour search.
XIX.	Inguinal.	12 „	......	Not made.
XX.	Inguinal.	1 „	......	Negative. ½ hour search.

the number of parasites, particularly of the small younger forms; that degenerating forms are found, and that in these cases the diagnosis by microscope is more tedious.

7. *Gland Puncture.*—Diagnosis by gland puncture would of course be

much preferable to excision of the gland in several respects, if equally trustworthy. For one reason or another the attempt was not made in seven cases. In three it was not successful, not sufficient fluid being obtained to make a smear. Of those in which fluid was obtained the parasite was demonstrated in four and not found in six. In thirteen attempts nine were failures, and only 31 per cent. succeeded. This is accounted for by the fact that in most cases the enlargement was not marked, and in children especially the mass of the gland was not sufficient to make it easy to bury the point of the needle with any certainty in it. In a few cases it was quite easy. In cases where the parasite was found quickly in the smears from an excised gland, the search was not continued very long in those from a puncture ; or possibly in several of the negative punctures results might have been reversed. In the four that gave positive results the average search occupied 18·5 minutes. It will be noted that in cases XII. and XVII. the puncture method was more successful than excision. In case XII. a two-hour search failed to show the parasite in the excised gland, though a more recent examination revealed it.

Further experience may modify these results and put puncture in a more favourable position, but at present it would seem that the excision method is far more certain and well worth the difference in initial trouble.

Objections to the Method.—The only possible objection to it is that it is an "operation" and likely to be refused as such by many patients. A little experience has shown that this has been less of a hindrance than one would have expected. Few of those who have been asked to submit to it have objected. The operation has always been performed under local anæsthesia ; the incision is small (less than an inch), and the whole affair, including the insertion of a couple of stitches, only takes a few minutes. More objection is often made to the hypodermic which introduces the anæsthetic than to the operation itself ; and the fact that in my series one patient was five years old, one four, and five others were thirteen or less, shows that the operation is not much of an ordeal. Three patients had a gland excised on more than one occasion, but did not object to the second incision. The wounds healed cleanly and without any annoyance, except in the case of three patients. One was a malingerer, who purposely tore open the incision ; and in one case there was a hæmorrhage into the wound, followed by sluggish healing, on both occasions when a gland was removed. The third has given very little trouble, and, indeed, the wound is a trifling one. The excision has been done by one of our Chinese staff in all but the first two cases, which I did myself.

Advantages.—1st, freedom from the danger which many of the best authorities believe attaches to visceral punctures. 2nd, ease and certainty with which the parasite is found. Of course this is only a short series of cases, and further experience here and elsewhere may be disappointing; but in case other observers have the same experience I have had, it would certainly seem to be a far quicker and a more certain method than that of splenic puncture, which, as just quoted, necessitates an average of four hours in the hands of an experienced observer like Bousfield, and fails altogether in a very considerable proportion of the cases. 3rd, the amount of material obtained. One gland will place at the examiner's disposal an unlimited number of slides, and leave available an adequate amount of infectious material for animal experiment and cultures.

Even granting these advantages, the diagnosis is still not as quick or certain as that of malaria or relapsing fever, and it may be that the solution will be a combination of gland excision and cultures from the material obtained. I have not been able to try this for the lack of an incubator.

Technic.

The excised gland is simply cut in two and smears made from the cut surface avoiding, as far as possible, contaminating them by blood. They are stained with any of the Romanowsky stains, and we have found it best to use a longer treatment than for malaria parasites, the pure stain being left on for five minutes, and the stain and water for ten. I have had the best results from Burroughs and Wellcome "Eosin-azur" soloids, made up as follows:—One soloid is dissolved in 10 c.c. of pure methyl alcohol and filtered. The staining is carried on just as with the Leishman stain; but I have thought the slides much less likely to have dirty precipitates in warm weather, and the colours are particularly bright, with strong contrasts between blue, red, orange, and purple elements. When staining for several minutes I put the slide on the cover of a Petri dish, and cover with the smaller part to prevent evaporation.

Treatment.

There is very little that is satisfactory to report. Of the series only ten stayed in the ward long enough to allow observation of the effect of remedies.

I. *Ipecacuanha.*—Following the suggestion of a fellow practitioner, who had seen remarkable improvement follow the administration of ipecacuanha, a thorough trial was given to that drug. In five cases no favourable result whatsoever was observed.

II. *Salvarsan.*—This was administered to nine patients, who received fifteen doses in all. There was little, if any, favourable effect, and what small improvement occurred was about what one would expect from the improved surroundings in the hospital. Cases without treatment, or with simple tonics, improved to about the same extent. In both cases examined on leaving the hospital the leucocyte count was not appreciably improved, and in one of them the parasites were more easily found than on admission.

III. *Quinine.*—This seemed to be of use in reducing the fever, but we have not been able to try it long enough in any one case to speak of permanent results. Its administration in such large amounts as seems necessary, and for such long times, is hard for the patient to endure; it is often vomited, and it causes anorexia. We have had the best results from gelatin capsules, and with intramuscular injections of the bihydrochloride.

Prognosis.

On this it is impossible to speak from first-hand knowledge. Judging from the answers to enquiries made of patients suffering from the disease, it is very fatal, as many deaths are reported to us and few recoveries. Of course, even some of those may not be Kala-azar. We have had only one death in our wards, but we have seen many patients in such condition as to leave little hope of their recovery. As already mentioned, in twenty-eight cases only two had lasted three years.

Cases.

Of a large part of the twenty cases reported, I have only the briefest possible record. In a number I simply had the opportunity to make an examination of the smears of an excised gland, as in cases IV., V., VI., XIV., and XIX. In a number of others the patients were prevented, for one reason or another, from coming into the hospital for observation and treatment. As the results of the examination of the blood and lymphatic glands are given in tabular form elsewhere they are omitted from the following synopsis. All were male cases.

I. Admitted Dec. 21st, 1910 : age, 4. Father died of the disease. Onset, 22 months ago, with fever and splenic enlargement. Fever irregular. Has had an irregular diarrhœa since August. Bleeding from nose, gums, and rectum. Has a prolapsus of rectum. Spleen fills left side of abdomen. Dec. 27th, spleen puncture negative, too large an amount of blood being withdrawn. Dec. 28th, puncture repeated ; two hours' search showed a leucocyte full of parasites. Dec. 30th, smears made from cut surface of post-cervical gland showed parasites in enormous numbers. Treatment: quinine. Went home Jan. 17th ; condition unchanged.

II. Admitted Feb. 22nd, 1911 ; age, 8. Two or three other cases in same village. Onset, 13 months ago, with fever. Spleen began to enlarge a month later. Has had diarrhœa with mucous in stools. March 4th, received salvarsan gm. ·2 in gluteal muscle. No reaction. While in hospital had 4 or 5 loose movements a day. March 10th, went home slightly improved.

III. Admitted Feb. 27th, 1911 ; age, 13. Onset, in June last, with irregular fever followed by splenic enlargement and occasional diarrhœa. Has had epistaxis and bleeding from gums. March 2nd, smears made from a puncture of a gland gave a negative result ; but those made from an excised gland showed the parasite in 25 minutes. On March 4th, received salvarsan gm. ·2 in gluteal muscle. For two days following he had considerable local pain and tenderness around injection, and for six days marked tenderness of the abdomen. Went home March 29th, "somewhat improved."

Cases IV., V., and VI. not treated.

VII. Admitted March 15th, 1912 ; age, 13. Onset, June or July the year before last, with fever, followed in ten days or so by enlargement of spleen. The febrile attacks came twice a day. From onset to admission he has had constant diarrhœa, ten or more stools a day. On admission was anæmic and cachectic. Abdomen distended, spleen almost to navel.

Ipecac was first tried, grains 20 every night, given as is usual in dysentery, and this treatment was continued from April 18th to 30th without any apparent benefit. On April 2nd, weight was 49 lbs. On the 4th he received salvarsan, gm. ·2 in the gluteal muscle. Little reaction followed the injection. His condition improved somewhat, the abdomen being more relaxed and the spleen softer. The diarrhœa continued, however, and was not in the least checked, while under observation, by other measures directed against it, such as ipecacuanha, bismuth, astringents, and rectal irrigations. April 18th, weight 49 lbs. ; condition rather worse. Salvarsan, gm. ·2, by vein. Following injection patient continued to get worse gradually, except that the spleen diminished slightly in size. He was given quinine, but without evident result. Went home on April 28th in worse condition than on admission.

VIII. Admitted March 16th, 1912 ; age, 13. Father and two uncles died of this disease. Onset, a year ago, with fever, followed in 10–20 days by splenic enlargement. Frequent epistaxis ; general condition fair ; spleen only moderately enlarged. Gland-smears show enormous numbers of parasites. Ipecacuanha was first tried, as in case VII., without any apparent good result. Weight, April 18th, 73 lbs. Has had cough and slight hemoptyses. Salvarsan, gm. ·2, by vein. April 24th, spleen slightly swollen. Following salvarsan had marked febrile reaction, with malaise, prostration, and anorexia. April 27th to May 3rd, received quinine, grains 35, by hypodermic injection, daily. May 10th improving ; weight 66 lbs. May 31st, again had fever and malaise. For six days was given quinine, grains 20, by hypodermic injection twice a day. Following this till June 12th, received grains 30, by capsules. June 12th ; improved. Spleen smaller ; weight 67 lbs. Salvarsan, gm. ·2, by vein. June 29th : red cells, 3,152,000 ; leucocytes, 2,560 ; large mononuclears, 41 per cent.; lymphocytes, 16 per cent.; polynuclears, 43 per cent. Spleen larger than on admission ; general condition about the same. Smears of an excised gland still contain numerous parasites. On the whole there is no evidence of any improvement.

IX. Admitted March 25th, 1912 ; age, 35. Several other cases in same village. Onset, nine months ago, in several attacks of fever lasting a week each, accompanied by epistaxis, and followed in a month from onset by enlargement of spleen. Is troubled with hoarseness ; urine has heavy trace of albumen. On admission spleen reaches navel.

From admission to April 4th, received ipecacuanha grains 30, nightly, without apparent result. From March 29th to April 18th, and from April 10th to April 18th, quinine, grains 15, t. i. d. April 2nd : weight 129 lbs. Salvarsan, gm. ·4, intramuscularly. April 18th, condition much the same. Weight, 128 lbs. Salvarsan, gm. ·24, by vein. April 26th, little change, though spleen is a little smaller. April 29th, went home.

X. Admitted April 1st, 1912 ; age, 32. In same village there are three or four cases. Invasion last March. Has had three irregular attacks of fever in a year ; had two rises of temperature a day. Spleen was not noticed till December. Has had severe epistaxis. Spleen reaches to left anterior sup. iliac spine. From April 2nd to 18th ipecacuanha, grains 30, nightly, but without effect. April 18th, weight, 140 lbs. Salvarsan, gm. ·4, by vein. Had a heavy chill following injection. May 10th, general condition improved. Spleen smaller and softer ; but weight has fallen to 134 lbs. May 15th, Salvarsan, gm. ·28, by vein. During part of this period received quinine, grains 40 a day, in solution. May 22nd, went home. Spleen much smaller ; general condition somewhat improved, but result of salvarsan not striking.

XI. Admitted April 13th, 1912 ; age, 28. Two nephews in another house have disease. History began 26 months ago with fever, spleen enlarging soon after. On admission spleen reaches almost to navel. April 13th to 18th, received ipecacuanha, grains 30, every night, but with no result. April 18th, weight, 100 lbs. Salvarsan, gm. ·35, by vein ; had a slight chill following it. April 25th, started on quinine, grains 15, t. i. d. April 26th, went home not improved.

XII. Admitted April 19th, 1912 ; age, 25. Has been ill since November with irregular fever, often having two attacks a day. Spleen enlarged about the time fever began. Has had epistaxis. On admission edge of spleen was 2 inches from navel. Soon after admission had a rise of temperature with anorexia, malaise, and prostration. On May 9th, gland puncture showed parasites, though they were not found in smears from an excised gland.

From May 9th to 15th, quinine, grains 60, was given by hypodermic injection daily, and fever gradually lowered. The quinine was then continued by mouth. May 15th, salvarsan, gm. ·28, by vein. June 3rd, has lost ground during fever, the spleen enlarging to navel. Weight, 113 lbs. Salvarsan, gm. ·27, by vein. June 12th, weight, 116 lbs. Salvarsan, gm. ·38 by vein. There was some local reaction at site of injection. June 22nd, general condition about the same. June 24th, white cells, 1444. June 28th, parasites readily found in three minutes' search from excised gland. July 1st, quinine in dilute sulphuric acid, intramuscularly. On leaving hospital, general condition somewhat better ; spleen just reaches navel. White cells, however, are about the same per c.mm., and parasites more numerous than on admission.

Cases XIII., XIV., and XV. not treated.

XVI. Admitted May 18th, 1912 ; age, 5. A neighbour has the disease. A month after the death of this patient two brothers came for treatment for Kala-azar. Patient has been ill a year with irregular fever. Has had epistaxis. On admission general condition very poor. On the inside of upper lip there is a gangrenous patch about ½ inch diameter. Under treatment the local condition improved, but the general condition got worse, and the patient died on the 21st. Though so ill and in the terminal stage, gland-smears only showed the parasites after 135 minutes' search.

XVII. Admitted May 22nd, 1912 ; age, 23. Only treatment was tincture of nux vomica for twenty days in hospital. During that time spleen diminished slightly in size, and general condition improved a little. In other words, he showed the same moderate improvement under hospital conditions, but with very little medication that other patients did under vigorous treatment.

XVIII. Admitted May 23rd, 1912 ; age, 25. Ill six months. Onset, with irregular daily fever, followed by splenic enlargement. Has a leg ulcer. Spleen enlarged two inches to right of navel. May 27th to June 12th, tincture of nux vomica. Condition improved somewhat. Spleen only reaches to navel. There was an oozing of blood into the wound when gland was removed, and union was delayed. June 12th, salvarsan, gm. ·5, by vein. A course of quinine by mouth was now begun. June 28th, red cells, 2,984,000 ; leucocytes, 1432 (compare figures on admission, reds, 2,976,000 ; whites, 1352). Parasites found in gland-smears more easily than on admission. July 4th, went home slightly improved.

Cases XIX. and XX. not treated.

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ON THE OCCURRENCE OF AN EPIDEMIC OF RELAPSING FEVER IN BULHAR, BRITISH SOMALILAND.

BY R. E. DRAKE-BROCKMAN, M.R.C.S., L.R.C.P.

As there appears to be no record of the occurrence of Relapsing Fever in Somaliland, a few remarks on an outbreak during the months of February, March, and April, 1913, will be of interest.

The town of Bulhar, where the epidemic took place, is not more than forty years old and is situated on the coast.

It consists of two portions, one, the town proper, consisting of mud and stone houses, while the other and larger part consists of many hundreds of huts arranged together in irregular blocks, each block belonging to a distinct tribe.

I have given a rough description of the town as the epidemic started in one of these collections of huts, commonly known locally as a "haffa."

The infected haffa is occupied by a race of outcasts called Midgans, who are chiefly engaged in sweeping the town or skinning the animals slaughtered daily for the consumption of the inhabitants. The Midgan haffa is consequently set somewhat apart from the other haffas and near to the slaughter houses and on the outskirts of the town. Being outcasts the Midgans are dirtier in their habits than the other Somalis, but, notwithstanding this, there is one haffa, the Dulbahanta, mostly composed of destitute people, adjoining the Midgan haffa, while several other haffas are within a short distance.

History of the Epidemic.

According to the patients themselves and the other inhabitants of the haffa, this particular type of fever has never before visited their or any other haffa in the town. The disease has been more common in the Midgan haffa than in the adjoining Dulbahanta haffa, while no cases have been reported from the other haffas in the town.

In the Midgan haffa there are 46 huts with 108 inhabitants; out of these on April 7th no fewer than 38 had had the fever and 17 were then down with it. In the Dulbahanta haffa, with 75 huts and 152 inhabitants, 6 had recovered, while 8 still had the disease.

On the appearance of the disease, a collection of the ticks found in the huts of the infected, or within a short distance of them, were made, and the following were the species obtained :—

I. Ixodinæ.

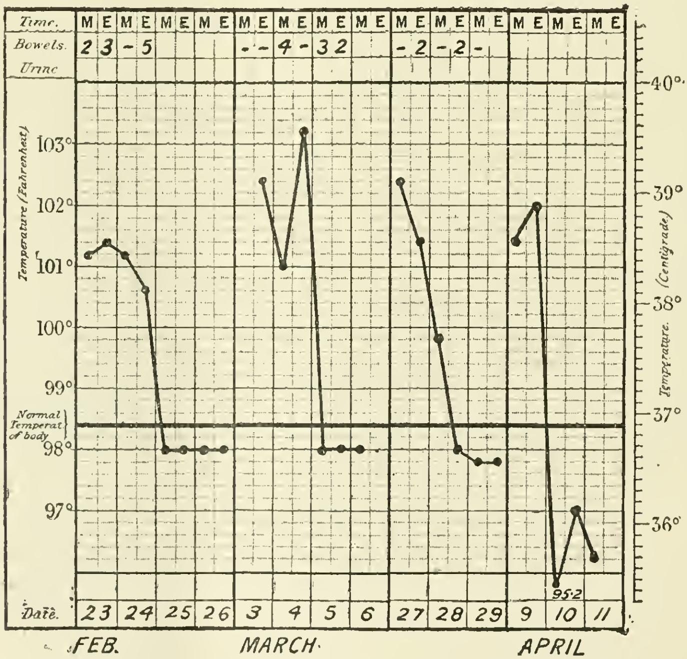
(a) *Hyalomma*	*H. ægyptium.*
(b) *Rhipicephalus*	*R. pulchellus.*
	R. simus.
	R. sanguineus.
(c) *Boophilus*	*Boophilus* sp.

II. Argasinæ.

(a) *Ornithodorus*	*O. savignyi.*
(b) *Argas*	*A. persicus.*

Ornithodorus savignyi was found almost entirely in the soft soil or sand covering the floor of the huts or immediately outside them. According to the inhabitants of the haffa it is very well known to them and is very common in all the coast towns. The Somalis have a special name for it, namely, Kūdkūdeh or Kūdkūda, whereas all the other ticks are called Shillin. Its bite is said to set up great irritation, and with subsequent scratching abrasions result which not infrequently become infected and ulcerate.

Chart I.



Argus persicus was common enough in the huts of those who kept fowls. In the daytime they had to be taken out of the cracks and crevices in the perches on which the fowls roosted at night. Every fowl examined showed

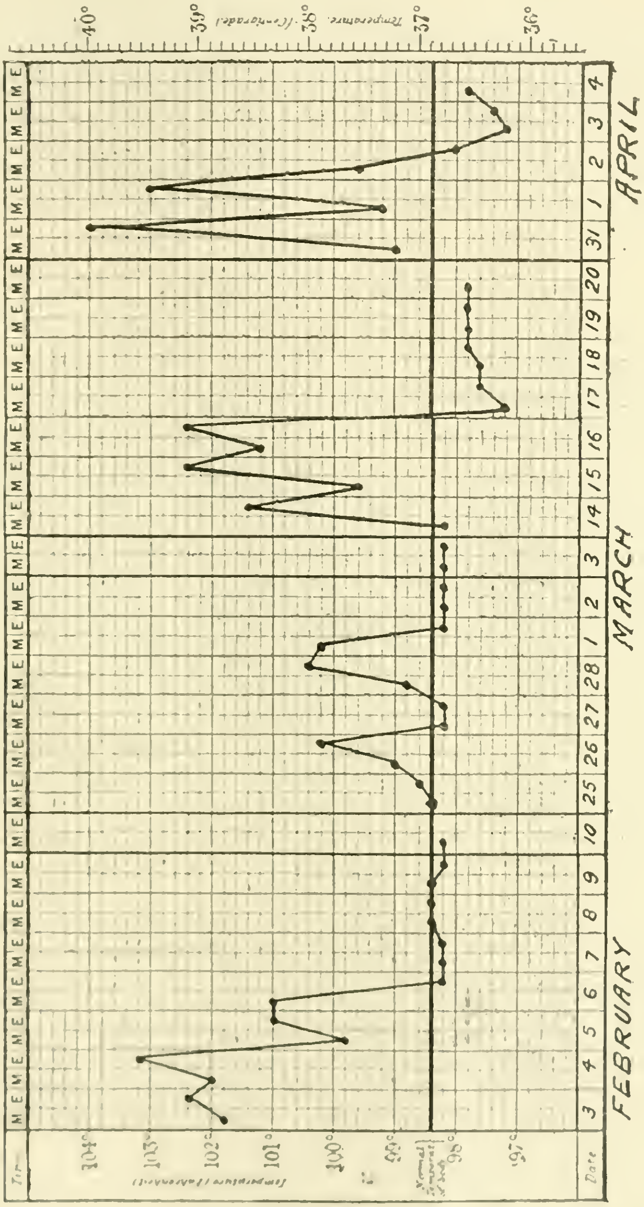


CHART II.

dozens of the minute larvæ fixed on to the skin around the neck and on the back.

The Somalis say it never attacks them and they have no name for it.

Blood Examination.

In the two cases, the temperature charts of which are shown, the blood was only examined somewhat hastily in Case II. during the initial paroxysm, while in both cases it was thoroughly examined during the relapses but without result. No spirochætes were found in the peripheral circulation in either case. The blood, however, of a large number of men, women, and children, suffering from the fever, who refused to remain as in-patients, was examined, and spirochætes were found in nearly every case during the initial paroxysm, but in not a single case were they seen during the relapses. They must have been either very scarce or not in the peripheral circulation at all; I am inclined to think the latter, as a very careful search was made for them in every case.

Species of the Spirochæta.

As regards the species to which the spirochæte belongs, everything points to the disease having been recently imported from British East Africa and the parasite being *S. duttoni*. During the last few years a very large colony of Somali traders has been formed in Nairobi, and seldom a week passes without a number of them arriving or departing from the Somali coast. The Somalis are probably correct when they say that, although the *Ornithodorus savignyi* has been known to them from their childhood, relapsing fever is quite new to them and could only have been imported this year.

On the first day of their appearance in the peripheral circulation, the spirochætes were usually seen lying in straight spirals, frequently end to end; later on during the remaining days of the paroxysm the spirals appeared to have opened out somewhat and were seen to be bent on themselves or coiled up or in some other irregular formation.

The spirochætes were generally first seen on the second day of the initial paroxysm, although in some cases they were not seen until the third day. They appeared to be equally plentiful when the temperature was high and when it had sunk to subnormal, provided the paroxysm was still on. At the end of the paroxysm they disappeared altogether from the peripheral circulation.

Symptoms.

The type appears to be the usual African type, only in these two cases which were kept under observation, there seems to be a certain regularity with regard to the relapses. It will be seen from the two temperature charts that the apyretic intervals are either of 6, 11–13, or 19–21 days' duration if we allow for the fact that the initial rise in the temperature in the third relapse in Case I. took place, as the patient says, on March 30th and the temperature rose in the second relapse in Case II., according to the patient, on the evening of the 26th. Thus, roughly speaking, the disease in both cases relapsed either at the end of one, two, or three weeks.

Unfortunately, it was found impossible to keep these two patients in the Hospital during the apyretic intervals, as not only did they themselves object, but, being Conservancy sweepers, their work had to be done.

The fever during the relapses, in both cases, appears to have been as severe as in the initial paroxysm and of the same duration, although in Case I. it was twice as severe.

As regards the other symptoms there was no difference in their severity during the initial paroxysm and the relapses. There was very severe headache with giddiness, vomiting, and profuse sweating. Constipation was marked in both cases both during the initial paroxysm and the relapses.

In one case, which, unfortunately, I was only able to examine once, there was photophobia, conjunctivitis, and iritis after the first paroxysm, while in two others there was bronchitis.

CASES OF SYPHILITIC PYREXIA.

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SYPHILIS as a cause of continued or irregular pyrexia simulating Tropical Fevers is well known to syphilologists but is often overlooked both in general practice and in the Tropics.

The importance now attached to the so-called Wasserman reaction and other methods of complement fixation will in future render the mistake less frequent, and in the supplementary course at the School these methods will be fully taught.

The reactions are not specific and are given by other diseases occurring in the Tropics, such as Trypanosomiasis and Tubercular Leprosy, but little confusion is likely to result as these diseases can be distinguished otherwise. In previous numbers we have illustrated the confusion that may be caused by general amœbic infection—Amœbiasis. In this we propose to illustrate the difficulties by cases that have occurred in the Albert Dock Hospital.

In these cases the effects of anti-syphilitic treatment by Potassium Iodide are illustrated, as that drug is practically specific for syphilis. Salvarsan and Neosalvarsan would no doubt give even more striking results, but as these drugs are specific rather for any spirochætal infection than for infections of *Spirochæta pallida* only, we prefer to illustrate only the effects of Potassium Iodide in cases that have been believed to be suffering from some "Tropical" disease.

Case 1.—J. K. C., Army Officer, aged 41, was admitted to Hospital on May 20th, 1913, complaining of malaria. The patient had been in West Africa and West Indies for the past six years, and stated that he had had attacks of fever from time to time during that period; and also noted that he had gradually been losing weight, so that now he only weighed 9 stone 8 pounds, as compared with his former weight of 11 stone.

Patient denied that he had ever had dysentery, blackwater fever, or venereal disease.

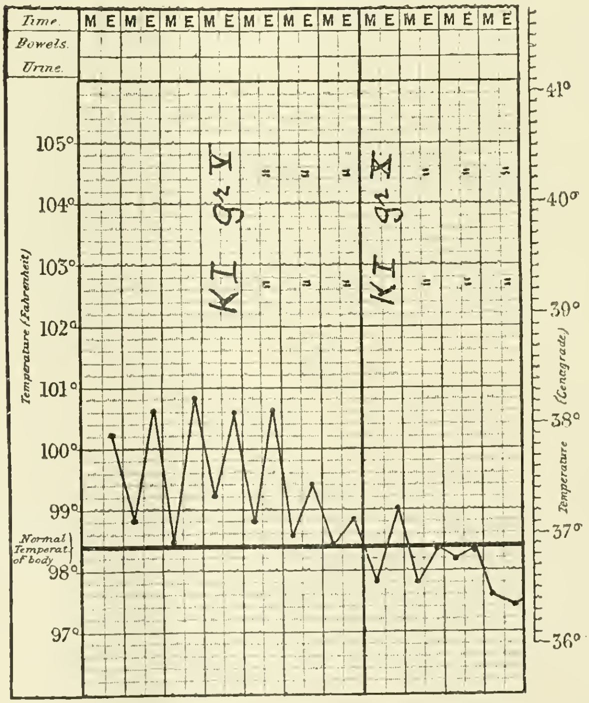
His present attack of illness started in March last with pain in the head, fever and marked sweating, but no rigors. His blood was examined, but no malaria parasites were found. He was treated for some time vigorously with quinine (gr. x. t.d.s.). This seemed to clear up the pain in the head, but fever still persisted and some pain was complained of in the legs.

On admission patient looked ill and showed a considerable degree of anæmia.

On examination the heart and lungs were found to be healthy. Nothing abnormal was to be detected in the abdominal region. The right leg was found to be painful over the internal semilunar cartilage and the edge of the tibia, but no redness or swelling was to be seen. Some pain also was complained of in this leg in the thigh and also down the sciatic nerve. In the left leg pain was complained of along the edge of the tibia only. In none of these situations were there any signs of inflammatory swelling. Examination of the blood yielded the following results :—

Red cells.	4,400,000	per c.mm.
White cells	5,000	,,
Hæmoglobin	85	per cent.
Lymphocytes	10	,,
Large mononuclears . .	15	,,
Polymorphonuclears . .	73	,,
Eosinophiles	2	,,

CHART I.



No parasites were present in the blood, and an examination of the fæces showed no parasites therein.

A glance at his temperature chart (Chart I.) shows that he had a rise to

100°·4 F. or thereabouts every evening during the first few days of his stay in hospital, the temperature reverting to about normal in the daytime.

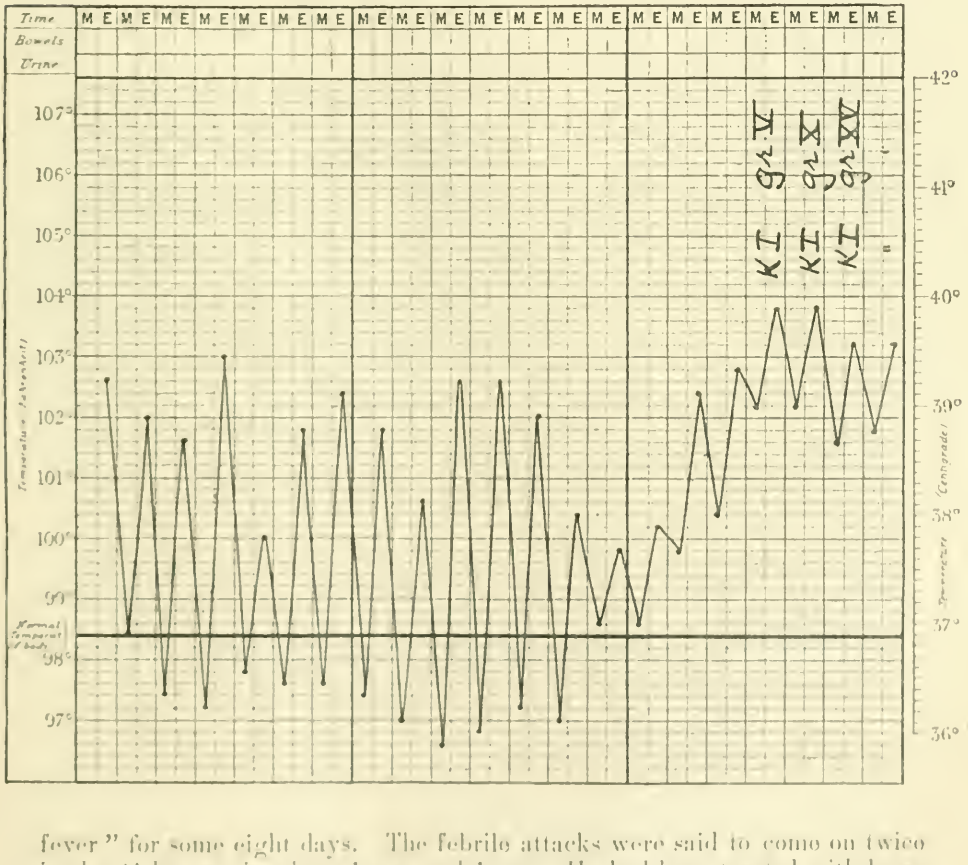
On May 23rd, four days after admission, he was placed on a course of treatment with Iodide of Potassium, commencing with grs. 5 t.d.s. and increased to grs. 10 t.d.s. on the 27th of the month.

The effects of this treatment were markedly beneficial, the nightly rise of temperature gradually declining, so that by the 29th of the month the normal was reached, and the temperature chart thereafter showed no rise above the normal either by day or night.

At the same time, the pains in the legs rapidly disappeared and he left the hospital on June 9th looking extremely well and expressing himself as feeling better than he had done for months.

Case 2.—A. A., aged 22 years, a lascar, was admitted to Hospital August 29th, 1912, with a history of having been suffering from "acute malarial fever" for some eight days. The febrile attacks were said to come on twice in the 24 hours, viz. about 1 A.M. and 1 P.M. He had been treated with large doses of quinine, which had failed to control the temperature. Owing to the

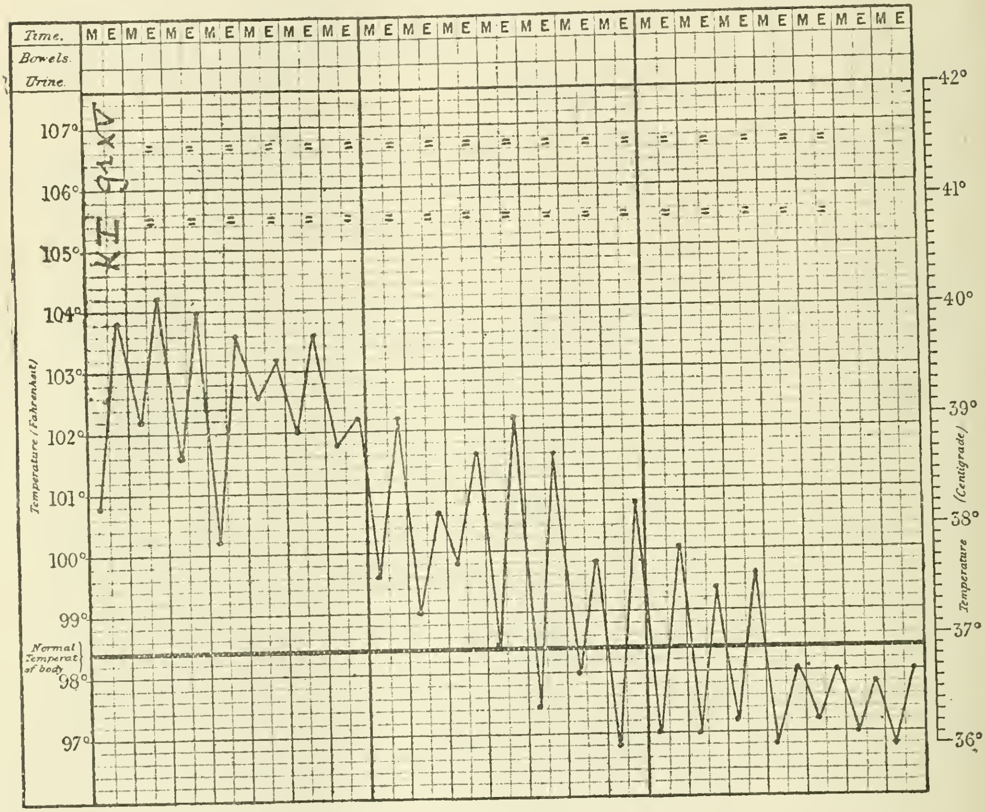
CHART II.



patient not being able to talk English it was impossible to get a comprehensive history.

On admission he was found to be a poorly nourished and anæmic looking man. He complained of no pain, but had slight cough with mucoid expectoration. On examination the lungs appeared to be quite healthy, except for a few scattered rhonchi. Heart was normal and no murmur heard. The spleen was slightly enlarged and just palpable below the costal margin. Liver and kidneys normal. The fundus of each eye was examined, but no abnormalities detected. No signs of syphilis detected.

Chart III.



Examination of his blood was made and yielded the following figures :—

Red cells.	4,000,000 per c.mm.
White cells	9,000 ,,
Hæmoglobin	100 per cent.
Lymphocytes	31 ,,
Large mononuclears . .	14 ,,
Polymorphonuclears . .	55 ,,
Eosinophiles	—

No parasites were detected.

Examination of the fæces showed no evidence of parasites.

The temperature chart for the first fortnight of his stay in hospital showed an intermittent fever, the temperature rising to about 103° F. about 10 P.M. every night, and falling to normal or below in the morning (*vide* chart II.). For the next two weeks the temperature became remittent, ranging between 100° F. and 104° F., but gradually regained the intermittent type in the middle of the fifth week (*vide* chart III.). In order to exclude the possibility of the disease being enteric or undulant fever, agglutination reactions were tried on several occasions but invariably proved negative. The sputum also was examined on many occasions, but no evidence of tubercle was obtained.

On September 16th (*i. e.* in the middle of the third week of illness) the patient was placed on a course of Iodide of Potassium, commencing with grs. 5 t.d.s., the dose being increased in the course of a few days to grs. 15 t.d.s.

At first this did not seem to produce much effect on the temperature, but by September 26th signs were becoming apparent of the temperature declining, and by October 6th it reached normal and remained so thereafter. On October 14th it was noted that the patient's temperature had been normal for nine days, and that he had manifestly put on flesh rapidly and looked very much better. He left hospital on October 30th quite well.

Case 3.—D. E., a cattleman, aged 35 years, was admitted to hospital on December 6, 1911, complaining of pains in the head, neck, and spine. He was brought in from his ship with his mind in a very weak and irritable condition, as if suffering from the after effects of alcohol, and it was impossible to get a clear history of his illness from him.

On examination, his temperature was 101° F., pulse 60, respirations 18. Pupils equal and active and fundi normal. Heart and lungs normal. Abdominal organs healthy.

A differential count of his leucocytes showed :—

Lymphocytes	7	per cent.
Large mononuclears . .	14	,,
Polymorphonuclears . .	79	,,
Eosinophiles	—	

No parasites were detected in either the blood or fæces.

The temperature chart showed an intermittent type of fever occasionally becoming remittent with nocturnal rises.

On December 9th, three days after admission, fairly marked retraction of the head was noted, which lasted for some 5 to 6 days. The pupils and knee jerks were normal, but the man's mental condition was impaired, so that he was always trying to get out of bed and spat all over the bed and floor.

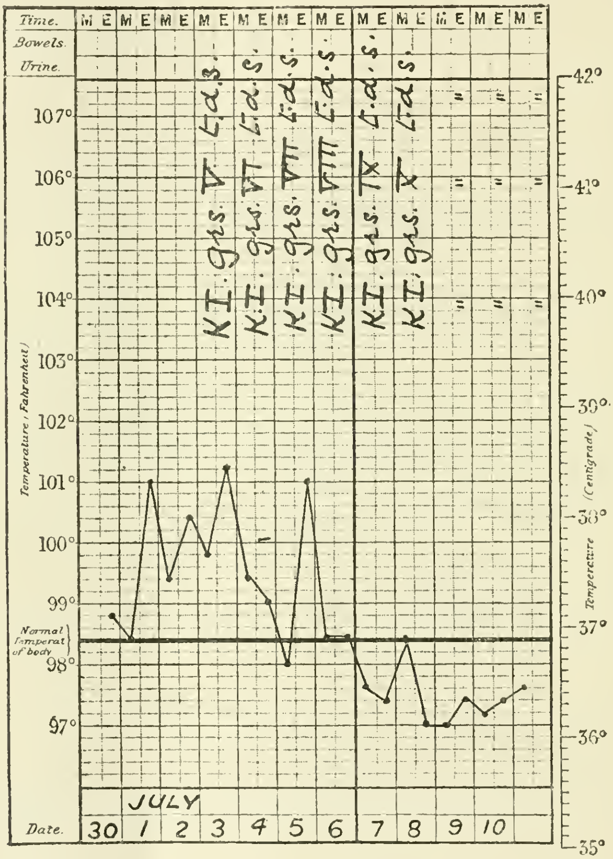
Lumbar puncture was performed, but examination of the fluid revealed no increase in leucocytes, nor any bacteria.

On December 10th he was placed on Iodide of Potassium, grs. x., t.d.s., but this was discontinued on December 14th owing to the patient's excited mental condition, which necessitated the administration of Hyoscine from time to time. The mental condition gradually improved and the temperature became less marked, but still showed rises above the normal.

On December 25th he was again placed on Iodide of Potassium, grs. 20 t.d.s. His temperature then rapidly dropped to normal, marked improvement in his general condition took place, and he left the hospital eight days later practically quite well.

Case 4.—S. Y., a Locomotive Superintendent, employed on railways in West Africa, was admitted to Hospital on June 30th, 1913, complaining of slight fever and shooting pains down the left side of the body, especially acute in the shoulder and over the ribs.

Chart IV.



Patient stated that on May 17th he had to go up the line during heavy rain and got a chill. On May 19th he saw a doctor, but had no fever then, only shooting pains down the right side commencing in the region of the liver and radiating towards the shoulder. He remained in much the same condition for a week when he complained of profuse perspiration lasting three days, followed by fever in which the temperature reached 103° F. This fever continued for two days and then subsided. He was then sent home, leaving the coast on June 14th.

On the voyage home he had more or less fever rising sometimes to 103° F., but coming down to normal on June 29th. During the voyage he took 15–20 grains of quinine daily.

On questioning him he admitted having dysentery in 1898, syphilis in 1905 (for which he was treated fairly thoroughly), and malaria from time to time but not during the last year.

On admission patient was a fairly healthy looking man, not markedly anæmic, and complained only of shooting pains in the left side. His temperature was raised to 98·8° F.

On examination nothing abnormal was detected in chest or abdomen.

Examination of the blood revealed no parasites, and no parasites or ova were to be found in the fæces.

A differential blood count yielded the following result :—

Lymphocytes	36	per cent.
Large mononuclears . .	8	„
Polymorphonuclears . .	54	„
Eosinophiles	2	„

On the day after admission temperature rose to 101° F. and remained raised above normal (*vide* chart IV.) with remissions till July 3rd. On that day he was placed on a course of Iodide of Potassium, commencing with grs. v., t.d.s. and gradually increasing the doses till grs. x., t.d.s. were administered.

On July 4th his temperature fell to below normal but rose again the next day to 101° F. On July 6th it again fell to normal, and thereafter no further fever was recorded and he was discharged on July 10th.

The shooting pains rapidly disappeared under the administration of Iodide, and the patient on leaving hospital expressed himself as feeling perfectly well.

LIVER ABSCESS, SINE PYREXIA.

J. W., a Foreman platelayer on the Sierra Leone Government Railway age 37.

Previous History.—Was 17 years in the army, during which he served for 3 years in South Africa during the war. Had no dysentery. Went to Sierra Leone in May 1909 and enjoyed good health during his first tour, except for eleven days, when he was ill with rheumatism.

Began his second tour on November 7th, 1910. On November 16th he was taken ill with pleurisy, necessitating his entering a nursing home, where he remained till November 24th. He then resumed work and kept well till February 6th, 1911, when he had to knock off work owing to an attack of diarrhœa with the passage of blood and mucus and also more or less continuous vomiting. On February 10th, 1911, he resumed work, though still suffering from diarrhœa and occasional vomiting with pain after food. Remained in much the same condition until March 11th, when he had to again give up work and returned to the nursing home. He remained there until March 30th. During the time he was in the home he suffered from pain after food, diarrhœa, and occasional vomiting. It was also noted that his weight, which was normally about 10 stone 8 lbs., had now dropped to 8 stone 6 lbs. He left the nursing home, practically unrelieved, on March 30th and was sent to a hill station, but no improvement took place, and he resumed duty on April 3rd. From April 3rd to April 27th he continued to work though still suffering from diarrhœa and pain in stomach, back, and shoulders.

On April 27th he was again sent to the nursing home, where the following note was made :—" Stools loose, dark coloured. Temperature 99. Says that when the diarrhœa stops he has pain in the abdomen and sometimes vomiting. Looks very emaciated. *Some enlargement of the liver.*"

On May 1st, 1911, the following note was recorded :—" Some tenderness in the epigastrium. Diarrhœa continues, bowels often moved but little passed. Rise of temperature to 100 yesterday. Subnormal this morning."

The patient was sent home on May 3rd, 1911, and says that during the voyage the diarrhœa was worse if anything.

He was admitted to hospital on May 22nd, 1911.

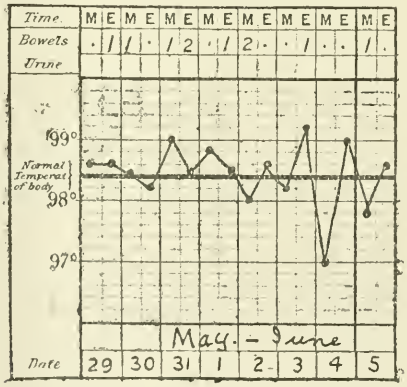
Condition on admission.—Patient very much wasted, the cheeks and temporal fossæ being much sunken.

Tongue moist and covered with a white fur. Abdomen distended. Skin of a muddy colour. Heart normal. Lungs normal, except for a few friction sounds in the right lung in the 7th and 8th spaces in the line of the lower angle of the scapula. Spleen not enlarged. Liver dulness extends from the 5th rib to the costal margin. Urine, 1020, no albumen or sugar.

The blood count on admission was :—

Total reds	3,080,000	
Total whites	17,000	
Hæmoglobin	75	per cent.
Lymphocytes	15	,, ,,
Large mononuclears	11	,, ,,
Polymorphonuclears	74	,, ,,
Eosinophiles	nil.	

No parasites present in the blood, but lamblia cysts detected in the fæces.



Patient was placed on milk, 3 pints per diem, increasing in a week gradually up to four pints. For the first three days patient had some seven stools a day of a greenish colour and offensive, with some streaks of blood, but after that the number of stools was about two a day.

On May 31st it was noted that liver was enlarged to two finger breadths below costal margin, but no tenderness was noted, nor did firm pressure with the fingers in the intercostal spaces over the liver elicit any pain.

Patient remained in much the same condition until June 6th, 1911, when he suddenly collapsed and died.

Post-mortem.—The body was that of an extremely emaciated man. On opening the thorax no pathological lesions were noted beyond some old pleuritic adhesions at the base of the right lung. In the abdomen all the organs were healthy except the intestines and liver. In the ileum the mucous membrane was distinctly thinned and degenerated. In spite of a most careful examination of the whole of the intestinal tract no evidence of dysenteric ulceration, either past or present, was obtainable.

The liver, of which the weight was 92 ounces, presented a fluctuating mass in the upper half of the right lobe, and on cutting into this a large quantity of pus of an "anchovy sauce" appearance escaped. There was a well-organised wall to the abscess cavity pointing to an old standing condition.

In the pus obtained from the abscess cavity living active amœbæ were obtained.

Observations on the Action on Trypanosomes of certain Drugs and of *Staphylococcus Pyogenes*.

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AND

J. W. SCOTT MACFIE, M.A. (Cantab.), M.B. (Edin.).

During the past year experiments have been carried out to show the toxic effects, if any, of certain drugs and of *Staphylococcus Pyogenes* on Trypanosomes, both *in vitro* and *in vivo*. Connor (1) reports three cases of plague that he treated with intravenous injections of tincture of iodine in dilute solution, all of which recovered. As he points out, the small number of cases obviates the drawing of any possible conclusions, but the results may at least be considered as inciting further investigation.

Davaine found that iodine destroyed the infectivity of septicæmic blood in a dilution of 1 in 10,000.

It seemed, therefore, a matter of interest to investigate the effects of iodine, not on a bacterial infection such as plague, but on a protozoal disease such as trypanosomiasis.

Bromine being of close chemical affinity to iodine was also chosen for experiment, as was osmic acid, from its rapid action in fixing protozoa in blood films.

Experiments in vitro.

A few initial experiments were made to determine the effects of dilute solutions of iodine, bromine, and osmic acid on trypanosomes in cover-slip preparations. For this purpose 1 in 10 solutions of the substances were prepared, and five minims of each were added to ten ounces of water.

A drop of each of these diluted solutions was then added to a drop of guinea-pig's blood containing trypanosomes on three cover-slips, and the effects watched under the microscope. The species of trypanosome employed was that for which one of us has recently suggested the name *T. nigeriense.* In each experiment a control preparation with saline solution was employed.

Only a few experiments were carried out, and the results were somewhat inconclusive, so that it will not be necessary to recount them in detail. As compared with the control, the immediate effect of iodine solution appeared to be a partial laking of the blood, and the trypanosomes tended to assume those atypical forms that have been described as "latent." After thirty minutes but few actively motile parasites remained, and an hour later nearly all the trypanosomes appeared to be dead. The action of the bromine solution was similar, but in some of the experiments it seemed to be transitory. After the first few minutes a good many of the trypanosomes had assumed a tadpole-like shape, but even two hours later there were still a number of actively motile parasites present. The action of the osmic acid solution was perhaps a little more intense, very few living trypanosomes being found in the preparations one hour after the beginning of the experiments.

Experiments in vivo.

The following solutions of the above drugs were made :—

Iodine	Tinct Iodi	m. vii.
	Aq. distil. ad . . .	ʒ i.
Bromine . . .	Brom. (2·5 p. c.) .	m. vii.
	Aq. distil. ad . . .	ʒ i.
Osmic Acid . .	Ac. Osmic (2 p. c.) .	m. xii.
	Aq. distil. ad . . .	ʒ i.

The trypanosomes employed were *T. vivax*, in a sheep, and *T. lewisi*, in rats. As intravenous injections in rats, frequently repeated, are all but impossible, it was decided to administer the drugs to them subcutaneously.

As a slight indication of the number of trypanosomes in the blood, it was decided to take the number observed relative to 100 leucocytes (in every case at least 200 leucocytes were counted), this percentage-ratio being called the "trypanosome index." This index was recognised to be quite inconstant, even under normal conditions, but seemed to be of considerable value as an indication when, if at all, a precise count by the Thoma-Zeiss method should be made, it being difficult to obtain a ready flow of blood daily, for a long period, from a small animal such as the rat.

Experiments with Iodine.

Exp. 1. Rat 41.

Index was 30·76 on the first day, when 20 minims of the solution described above were injected. As this dose appeared to have a marked effect on the rat, the future doses were halved, and 10 minims were injected daily until the 16th day inclusive.

The index remained high but very irregular, varying from 40 on the 13th day to 119 on the 9th ; but on the 11th day it was as low as 10·5. On this day only, as noted in a previous paper, marked auto-erythro-phagocytosis was observed.

On the 16th day, a slight skin slough commencing, and no effect on the trypanosomes being apparent, the experiment was stopped.

Exp. 2. Sheep 65.

One drachm of pure tincture of iodine was introduced into the external jugular vein of a sheep infected with *T. vivax*. On the 2nd day the sheep was obviously ill, trypanosomes remaining very numerous. Death occurred on the 5th day.

Post-mortem examination showed acute bryncho-pneumonia, with blood-stained pleuræ, a large mass of gelatinous lymph within the pericardium, over the apex of the heart, and intense engorgement of the kidneys.

Boehm and Berg have shown that large doses of iodine will produce œdema of the lung, hæmorrhagic exudation into the pleural cavity, and great renal congestion.

Experiment with Bromine.

Exp. 3. Rat 42.

First day, index 36. As in the first experiment, the initial dose of 20 minims being apparently too large, was followed by daily injections of 10 minims. In contrast with Rat 41, the index remained stationary during the injections, the greatest variation being from 22·5 on the 3rd day to 37·5 on the 6th.

The local effect, however, was more marked, a skin slough commencing the 7th day, whereupon the injections were stopped. The blood was examined daily until the 16th day, but nothing of interest was observed, except some leucocytosis very possibly due to the septic state of the skin.

Experiments with Osmic Acid.

Exp. 4. Rat 43.

Index on the first day was 147, when 10 minims of the solution were injected. On the 2nd day intense leucocytosis was present, with great destruction of leucocytes. Ten minims were again injected. Four hours later the index was 88·23, and there was marked phagocytosis of trypanosomes and red cells, to which further reference will be made. On the 3rd day 5 minims were injected. The blood was thick and tarry, leucocytes less disintegrated, phagocytosis of red cells still marked, but of trypanosomes less so. On the 4th day the rat was obviously dying. There was leucopenia ; trypanosomes were extremely numerous.

After death, a septic peritonitis was found to be present, some of the osmic acid having apparently entered the peritoneal cavity. The subcutaneous tissues at the points of inoculation were blackened and necrosed.

Exp. 5. Rat 47.

To lessen the local action of osmic acid the dose was halved, 5 minims of the solution being given daily, diluted with an equal volume of normal saline. The index, which was 32 on the 1st day, was exceedingly variable, but was not again as low as 32 except on the 3rd day, when it fell to 18·5. On this day only, as on the corresponding day of Rat 41, auto-erythro-phagocytosis was observed.

There was no other noticeable feature, except that on the 8th day there was some leucocytosis and considerable destruction of leucocytes.

The observations were stopped on the 16th day.

The Mechanism of the Destruction of Trypanosomes in the Body.

Opinion is divided as to the mechanism by which trypanosomes are disposed of in the body tissues. By adding trypanosomes to a hanging drop of eel's serum, Laveran and Pettit (2) obtained rapid trypanolysis by the serum. Massaglia (3) injected trypanosomes from guinea-pigs, mice, and rats into the peritoneal cavity of cold-blooded vertebrates. Examination of the peritoneal fluid, lymph, and blood showed trypanolysis, but in no case was phagocytosis observed. Delanoë (4) has described the phagocytosis of the Leptomonas of Kala-azar when injected into the peritoneal cavity of a mouse, and he has also seen phagocytosis of *T. lewisi*. Roudsky (5) has observed the phagocytosis of *T. lewisi* in the peritoneum after injection of immune mice, the trypanosome sticking to the leucocyte by its posterior end, round which the leucocyte sends a sleeve-like prolongation.

Delanoë (6) describes phagocytosis in detail. The trypanosomes are destroyed in full vitality, and if the flagellum happens to be the last portion ingested it may be seen to be motile until the moment of its disappearance.

In our own case the ingestion of trypanosomes was watched in the living blood. In one instance three trypanosomes were observed by the side of a phagocyte, and in spite of violent contortions they appeared unable to get away, although other trypanosomes were seen passing freely in the immediate neighbourhood. Gradually each was destroyed, and all were absorbed by the phagocytes within thirty minutes. In stained films we observed appearances similar to those figured by Brumpt in his 'Précis de Parasitologie,' 1910, p. 124.

The Toxic Effects of certain Bacteria on Trypanosomes.

Levaditi and Twort (7) investigated the action on trypanosomes *in vitro* of *B. coli*, *B. prodigiosus*, *B. mesentericus*, *B. pyocyaneus*, and *B. subtilis*. The first four had little or no effect; but when 15 drops of a *subtilis* culture were mixed with 2 drops of infected blood at 37° C., trypanolysis was complete in 25 minutes. This effect was proved to be due to a toxin, the filtrates of washed cultures being active, the washed bacilli themselves inactive. They found that the trypanotoxin was not effective in the living blood, possibly because it was seized first by the living cells of the blood.

Rodet, Mdlle. Rubenstein et Bader (8) investigated the effects produced by bacterial infections on the course of an infection with trypanosomes. The trypanosome used was *T. brucei*, and the bacteria were Eberth's bacillus, *B. anthracis*, *Staphylococcus*, and *Streptococcus*.

They found that animals infected with Nagana were more, rather than less, resistant to bacterial infections; in no case was a bacterial infection of curative value.

Thomas and Breinl (9) stated that guinea-pigs suffering from tuberculosis were more susceptible than healthy guinea-pigs to infection with trypanosomes, but gave no account of their experiments. Orsi (10), on the other hand, proved that the tubercle bacillus had a definitely antagonistic effect on the trypanosome of Nagana, a previous infection with tuberculosis completely preventing the appearance of the trypanosome in inoculated guinea-pigs. The effect was apparently due to the vital action of the bacillus, tuberculin, killed tubercle bacilli, and ethereal extracts all proving inactive.

Experiments with Staphylococcic Vaccine.

Whilst observations were being made on a rat normally infected with *T. lewisi* it was noticed that the rat showed staphylococci in its blood, and that the trypanosomes rapidly decreased in number and finally vanished, and that this was then followed by recovery of the rat from its staphylococcal infection.

To investigate whether this were any relation of cause and effect, blood was procured aseptically from the rat, cultures of staphylococci grown, and a vaccine was prepared.

Exp. 6. Rat 52.

First day, index 9. Injection of two million staphylococci. This was followed by leucocytosis and a great fall in the trypanosomes, so that an index was barely obtainable. On the 6th day, two millions injected ; there was a temporary rise in number of both leucocytes and trypanosomes on the 7th and 8th day.

On 14th day three and a half millions were injected ; there was a marked increase in the trypanosomes. Further injections of four millions each were given on the 20th, 26th, and 33rd days. The trypanosomes decreased, the index after the 26th day, when it was 4, never being above 1. On the 42nd day no trypanosomes could be found ; but one was seen on the 44th day, and on the 47th they were fairly numerous again.

Exp. 7. Rat 61.

First day, index 115·5 Three million staphylococci were injected on the 1st and 8th days, and four millions on the 11th, 17th, 23rd, and 30th days. There was no obvious effect. There was slight leucopenia, instead of the leucocytosis seen in Rat 52 ; the trypanosome index remained at a high level.

Exp. 8. Rat 62.

First day, index 82·5. Injection of four million staphylococci, which was repeated on the 8th day. Trypanosomes remained very numerous, and the rat died on the 9th day.

Conclusions.

Our results have been almost entirely of a negative character. Of the drugs experimented with, iodine and osmic acid appear to have a very disturbing effect, not only on the trypanosomes, but also unfortunately on the host.

We consider that there can be no doubt that trypanophagocytosis does occur, and that large numbers of trypanosomes may by this means be rapidly destroyed.

Experiment 6 might be construed as meaning that the trypanosomes were reduced by the effects of the staphylococcic vaccine to a vanishing point, and that, on stopping the vaccine before cure was complete, the trypanosomes returned.

However, the vaccine produced no similar effects in Experiments 7 and 8, and it is probable that the variation in the number of trypanosomes seen in Experiment 6 was merely the normal variation of *T. lewisi* over a prolonged period.

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AUTO-ERYTHROPHAGOCYTOSIS AS AN AID TO THE DIAGNOSIS OF TRYPANOSOMIASIS.

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(Preliminary Communication.)

AUTO-ERYTHROPHAGOCYTOSIS has been observed by Connal (1) in the blood in cases of trypanosomiasis, malaria, smallpox, filariasis, and ankylostomiasis, but in the last three it is possible, if not probable, that it was due to an added malaria.

Wright (2) has seen it in two cases of pneumococcal infection, and it has been seen *in vitro* by several observers—Barratt (3), Hektoen (4), and others.

Connal found erythrophages in the blood at the same time as, and in some cases after the disappearance of, trypanosomes, and he mentions one case that showed a single macrophage a month after inoculation, before the appearance of trypanosomes, but he does not state whether the cell contained erythrocytes, nor whether the animal—a guinea-pig—finally developed trypanosomiasis.

We have examined several animals infected with trypanosomes, and in some have noticed the ingestion of red cells. The erythrophages observed resemble those described by Connal. The number of red cells ingested by a single leucocyte varied from one to twelve or even more. Many phagocytes showed red cells partially absorbed, the depth of staining varying inversely with the state of absorption ; some showed apparent vacuoles which we consider may be the final stage ; many showed a large number of granules evidently derived from digested red cells—perhaps one possible source of the pigmented mononuclears so frequently seen in malaria. The shape of the erythrophage when containing cells is largely determined by the number of its red cells, one or two causing but slight alteration, ten or twelve causing enormous distention and a bloated appearance of the leucocyte. In a few instances a red cell appeared to be placed on the top of the centre of the nucleus.

The ratio of cells containing ingested reds to other leucocytes is, as a rule, very low, seldom over one per cent., but one case (Mouse 31) showed no less than 11·5 per cent. The variation in the percentage ratio of trypanosomes to leucocytes (which we will call for convenience the trypanosome index), whether erythrophages are present or not, appears to be inconstant, but we have several times noticed a drop in the index on the same day as erythrophagocytosis. For example, in Rat 41, from July 20th to July 26th, the index remained persistently high—58 to 119—except on July 25th, when it was 10·5, and in Rat 47, from July 24th to July 28th, the index varied from 86·5 to 173, except on July 25th, when it was 18·5. In each case the low index occurred on the only day on which erythrophagocytosis was observed.

The Infecting Trypanosome.

(1) *T. lewisi.* Connal failed to obtain ingestion of red cells in rats infected with *T .lewisi*, which he attributes to its non-pathogenicity ; we, however, have obtained marked phagocytosis of reds in certain rats that were undergoing treatment, probably owing to a relative rise in the pathogenicity of the trypanosomes.

The other trypanosomes we employed were (2) a trypanosome closely resembling *T. gambiense*, obtained from a case of sleeping sickness.

(3) A trypanosome resembling *T. duttoni*, from a mouse—naturally infected.

We have divided our cases into three groups :—

Group A. In which Leucocytes containing red cells were present in blood which later showed trypanosomes.

1. Monkey 37. Injected with Trypanosome (2).
   Fifth day; showed erythrophagocytosis.
   Seventh day; showed trypanosomes.
2. Mouse 31. Injected with Trypanosome (2).
   Eighth day; intense ingestion of reds.
   Ninth day; trypanosomes appeared, and increased in number whilst the ingestion decreased, until the thirteenth day, when the mouse was accidentally killed.
3. Goat 11. Injected with Trypanosome (2) on June 4th, showed ingestion of red blood cells on July 11th, but it was not until August 5th, twenty-two days later, that trypanosomes were sufficiently numerous to be detected in the peripheral blood.

Group B. In which both were at some time present simultaneously.

1. Rat 40. Injected with Trypanosome (3).
   Third day; a few trypanosomes.
   Fifth day; a few trypanosomes and a few ingested red cells.
   Sixth to twelfth days; no further trypanosomes, but a few scanty ingested reds.

2. Rat 41. Caught infected with *T. lewisi.*
   Ten days later scanty erythrophagocytosis was seen (Index 10·5).
3. Rat 43. Caught infected with *T. lewisi.*
   Second day; phagocytosis very marked.
   Fourth day; unfortunately died.
4. Rat 47. Caught infected with *T. lewisi.*
   Third day; ingestion of several reds (Index 18·5).
   Succeeding days; no further ingestion, though trypanosomes continued.
5. Mouse 30. Caught infected with trypanosome (3).
   Second day; one red seen ingested; a few trypanosomes.
   Fourth day; more reds phagocytosed; many trypanosomes seen.
   No further reds were seen phagocytosed; the mouse died on the tenth day.
6. Monkey 21. Inoculated with Trypanosome (2).
   Tenth day; Trypanosomes appeared.
   Eleventh day; Erythrophagocytosis also seen.

Group C. In which phagocytosed red cells were seen after the disappearance of trypanosomes.

1. Rat 40. Also falls under group B.

Relation to Protozoal Diseases.

We have also observed ingestion of red cells in one case of Blackwater fever. A guinea-pig injected with blood from a case of Yellow fever showed a rise of temperature on the second day, accompanied by erythrophagocytosis; the blood did not contain any parasites of the other diseases mentioned.

We have, however, so far failed to find this in blood from Yellow fever patients. Assuming that the causal organisms of these diseases are of a protozoal nature, as seems probable, there would appear to be a certain relationship between erythrophagocytosis and protozoal diseases such as those mentioned: Trypanosomiasis, Malaria, Blackwater fever, and Yellow fever.

Question of Immunity.

Connal suggests that the phenomenon is related to the development of immunity, but its occasionally initial appearance would seem to negative this theory, unless perhaps it were connected with an abortive attempt at immunisation.

Diagnostic Significance.

We put forward the suggestion that erythrophagocytosis may be of material aid in the diagnosis of trypanosomiasis.

In group A, which may be considered to correspond to early cases, the appearance of trypanosomes in the blood was definitely preceded by phagocytosis of red cells; in group C, or cases of a late stage, phagocytosis was seen after trypanosomes had disappeared.

Moreover, trypanosomes are not by any means invariably to be found in the blood in cases of trypanosomiasis. Trypanosome (2) was obtained from one of several cases met with in Nigeria, in which repeated blood-examinations were negative, though trypanosomes could be readily obtained from gland-juice or from cerebro-spinal fluid—methods, however, not always practicable with native patients. In such cases, it seems to us, the finding of erythrophagocytosis would be of considerable importance.

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Diagnosis and Treatment of Undulant Fever.—Bassett-Smith (Journ. of Hygiene, xii. 1913, No. 4, p. 497) for agglutination tests prefers the sedimentation method, for which an emulsion of a 48-hour old agar culture in physiological salt solution should be employed. Three dilutions of the serum are made, 1–40, 1–100, and 1–400, and the tubes are placed in the blood-heat incubator for two hours and the results noted. The tubes should then be allowed to stand at laboratory temperature and the results recorded after a further period of twelve hours. In some 2000 observations, only once was a positive agglutination obtained with a control serum. Complement-fixation tests may also be employed and are satisfactory. Absence of agglutination does not necessarily negative a diagnosis of Undulant fever: in cases of long duration it may be absent. Isolation of the organism from the blood is another method that may be used, but similarly may fail in long-standing cases. An organism, the *M. paramelitensis*, has been found by Nègre and Raynaud in certain cases of Undulant Fever. In such cases, the blood may not agglutinate the *M. melitensis* but does agglutinate the *M. paramelitensis*. A case of this kind is recorded by Bassett-Smith (Journ. of Trop. Med. & Hygiene, Feb. 15, 1913). As regards treatment, yeast or yeast-products has been found of service in the neuritis of the disease. Vaccines (100 to 500 millions) should be given every 5–7 days: it is contra-indicated when the pyrexia is continuous or remittent.—R. T. H.

Protective Inoculation against Cholera.—W. M. Haffkine has published, under the above title (W. Thacker & Co., 1913), a useful summary with bibliography of the whole subject of cholera preventive inoculations. After a survey of the principal biological features of the cholera vibrio, and a summary of methods for propagating the organism (Part I.), the problem of the immunisation of man against cholera is discussed (Part II.). Here the methods adopted and some of the results obtained in the earlier work on preventive inoculation against cholera by Haffkine and others are detailed. The vaccines employed were living ones, a first or weak (No. I.) vaccine sometimes being used before the second or strong (No. II.) vaccine. Part III. is devoted to a study of the effects and efficiency of the strong or No. II. vaccine after "devitalisation." The author considers that it is preferable to use the "devitalised vaccine" in its entirety, rather than to employ extractions or other derivations of it. He suggests that the devitalised vaccine may be prepared by two methods, (*a*) prepared by prolonged cultivation in a fluid medium and devitalised by heat and carbolic acid, (*b*) prepared by cultivation on a solid medium and devitalised, as soon as developed, by a solution of carbolic acid.—R. T. H.

V.—PUBLIC HEALTH.

PROTECTION OF THE MILK SUPPLY IN BRITISH GUIANA.

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THE control of the milk trade in Georgetown, the capital city of British Guiana, is an exceedingly difficult problem. The trade is mostly in the hands of small peasant farmers, usually of East Indian origin, but with a sprinkling of black people. Each man owns one or two cows, which are allowed to roam about the rough pastures and portions of abandoned estates in a practically wild state. The milking cows are rounded up about 4.0 A.M. and milked in a rough shed or even in the open pasture. After this the cows return to the pastures. The calves are mostly stall fed. The milk thus obtained is sent into the city in small cans ranging from 2 to 10 gallons' capacity, and either supplied direct to regular customers or hawked about the city until disposed of. The above holds good in a lesser degree for the villages and country districts.

The number of persons connected with the trade in a 12 mile radius of Georgetown, a city of roughly 60,000 people, is over 800, and the amount of milk entering the city daily is estimated at about 1500 gallons. From these figures it will be seen that the control of the milk supply is a very difficult task.

From time to time various Milk Regulations have been drawn up and published by the Municipal Authorities, but without any appreciable effect on the purity of the milk supplied.

In consequence of several cases of typhoid fever having been proved to be caused by the sale of infected milk, an investigation into the purity of the milk supply was recently carried out by the Government Public Health Department. A large number of samples were examined for the presence of fæcal bacilli, the investigation lasting over six months. It was found that milk containing as many as 100,000 fæcal organisms per c.c. was by no means uncommon, whilst 20 per cent. of the samples examined contained over 1,000,000 fæcal organisms per c.c. As a result of these investigations, the Milk By-Laws of 1912 were drawn up by the Local Government Board and strictly enforced. These By-Laws came into operation in January 1913, and an English sanitary inspector was obtained through the Royal Sanitary Institute to assist the Health Officers in carrying them out.

The By-Laws are based on a system of registration of the cattle pens where milking operations are carried out, the issuing of licences to milk vendors, with numbered tablets to attach to all cans, and a numbered badge to be worn on the left arm by all persons engaged in retailing milk.

SECT. 1 is as follows:—It shall not be lawful to keep a dairy, cattle byre, or cattle pen unless registered with the Local Authority of the district in which such dairy, cattle byre, or cattle pen is situated.

SECT. 2.—It shall not be lawful for any person to offer or carry milk for sale unless licensed by the Local Authority.

SECT. 3.—Every registration and every Licence expires on December 31st of the year of issue.

SECT. 4.—The Overseer of each Local Authority shall keep and from time to time revise and correct the register of all registrations and licences issued.

These four are the principal sections and the basis upon which the By-Laws are constructed.

Section 1 provides for the registration of the pen, and the conditions on which a certificate of registration can be obtained are stringent. Before a cattle pen or milking shed can be registered it must first be visited by an Inspecting Officer, of whom there are three only, appointed by the Government—the Govt. M. O. H., the Asst. Govt. M. O. H., and the Government Sanitary Inspector. The conditions attached to the issue of a certificate are:

1. The site must be approved of.
2. The pen must be constructed with a concrete, brick or tiled floor properly drained.
3. The walls (if any) to be constructed of impervious material to a height of four feet from the floor.
4. The roof must be of impervious material (corrugated iron usually) and means provided for water storage.
5. The pen is to be lime whited, with lime white to which is added 5 per cent. of carbolic acid, and re-whited every three months.
6. Efficient drainage must be provided to the nearest main drain, and a proper dungstead provided not less than ten feet from the milking shed.

If the cattle pen or milking shed complies with these requirements, and each animal is allowed 40 square feet of floor space with 300 cubic feet of air space, the Inspecting Officer then gives a certificate to that effect.

The owner of the pen then takes the certificate to the Overseer of the Local Authority, who enters the name of the owner, situation of the pen and number of animals for which the pen is constructed in the register, and then gives a certificate of registration. No charge whatever is made for these certificates.

This registration enables the pens to be kept under surveillance as regards cleanliness of shed and animals; and they are visited regularly by the Overseer and Inspecting Officer. The penalty for keeping an unlicensed cattle pen is $10 for the first offence and not exceeding $50 for a subsequent offence.

Section 2 provides for keeping under control the persons engaged in the milk trade, whether as milkers or sellers. The applicant for a licence to handle or sell milk must give the following particulars :—

1. His name, address, and the situation of the cattle pen whence the milk is obtained.
2. Furnish a certificate from a Government Medical Officer that he or she is free from infectious disease and fit to be so employed.

On production of this certificate, for which he is charged 24 cents by the Government Medical Officer, and having given the above particulars, the applicant's name is entered in the register of milk sellers for the district, and a licence to sell milk is given free of charge.

With the licence an arm-badge, and metal tablets to attach to the milk vessels in use, are also issued; these badges and tablets all bear a number corresponding with that of the licence and the year of issue. The charge made to the milk seller for the metal badge to be worn on the left arm is 12 cents, and a charge of 6 cents is made for each metal tablet for use on the milk vessels.

This section enables the Authorities to at once trace the source of any milk found, on chemical analysis, to be of bad quality or, on bacteriological analysis, to be infected or otherwise unfit for consumption.

By means of this licence system a recent outbreak of typhoid fever in the city was easily traced to an insanitary cattle pen and the washing of milk vessels in infected water. A reference to the register of licences at once reveals the source of the milk supplied by the milk vender under suspicion. Severe penalties are inflicted upon vendors detected in obtaining milk from sources other than those stated in their licences.

A number of samples are taken for analysis weekly by an attendant in the Laboratory. These are collected in sterilised bottles provided with labels. When a sample is taken, the letters, number, and date of the badge worn by the vendor are recorded on the bottle, which is returned at once to the Laboratory. A bacteriological analysis is then carried out at once, the total bacterial content being estimated, together with the number of faecal organisms present in 1 c.c. or, if absent in this quantity, present or absent in 2 c.c., 5 c.c., or 6 c.c. If the milk is shown to be very bad as regards faecal contamination, the pen from which the milk is obtained can be at once traced by reference to the register of licences, and the Sanitary Inspector is instructed to visit.

This method enables us to keep a fairly rigid control over the cleanliness of the cattle pens and animals. Since the people have been brought to realise that it is possible to detect insanitary surroundings in this manner, the analyses have shown a remarkable improvement. Samples taken at 10.0 A.M. formerly showed the presence of 100,000 or frequently 1,000,000 faecal organisms per c.c.; now it is unusual to get one showing 10,000 or 1000 faecal bacilli per c.c.; a fair number show 100 per c.c., whilst many samples examined are as low as 10 per c.c. or less, a truly remarkable improvement when the ignorance of the people engaged is considered. No doubt the knowledge that a constant check is kept on the milk retailed by the vendors has been largely instrumental in bringing about a great improvement in the sanitary condition of the cattle pens.

It is also invariably pointed out by the Inspecting Officer that unless the cans are scalded before use, it is hopeless to expect the milk to remain sweet and clean in a tropical climate. This appeals to the people, as it enables them to stay in town until their stock is sold out without fear of prosecution.

The question of supervising the cleanliness of the cows and milkers themselves is a much more difficult problem, involving, as it does, surprise visits to the milking sheds at 4.0 A.M., whilst the milking is in progress. This is done periodically in different districts, the visits being kept as secret as possible. In flagrant cases of this kind the magistrate is asked to inflict a severe penalty.

One of our greatest troubles is the provision of a pure water-supply for washing cans and milk vessels generally. At present the best we can do is to see that provision is made for storing rain-water, but this source is far from satisfactory, and the addition of chemical purificants such as chlorine objected to. The By-Laws provide for the scalding of milk vessels with boiling water before use, also for the washing of the hands of milkers and the cows' teats, but these regulations are very difficult to put into force, and it is only occasionally that offenders are detected by means of surprise visits, and penalties inflicted.

It is difficult to make a native who is accustomed to scraping up cowdung by hand, making it into cakes, to dry in the sun for use as fuel, understand that a can that has been merely rinsed out in a neighbouring trench is not clean enough. And as for washing the hands, together with the cows' teats, in soap and water before milking, this is looked upon as an absurdity that is to be avoided as often as possible.

Much depends on the Overseer of the district, and the best results are seen only where he is keen and interested in the work.

Lectures are given by the Health Officers on sanitation generally to village overseers and others interested in the work, and examinations held to test their knowledge in these subjects. Certificates are given to the successful candidates, and it is gratifying to record that these certificates are already much prized and sought for.

Experiments with the Micrococcus melitensis.

By C. H. MARSHALL, M.R.C.S., L.R.C.P.,
Uganda Medical Staff.

Dr. Eyre handed to me five strains of *M. melitensis* distinguished by the numbers 1–5 inclusive, obtained from the following sources:—

No. 1. Malta Milk.
No. 2. Bassett Smith.
No. 3. Human case (fatal), Richards.
No. 4. Bassett Smith.
No. 5. Malta Milk.

These I studied carefully as to their morphology and cultural characteristics, and made myself familiar with the life-history of *M. melitensis* under laboratory conditions.

Of the five strains, No. 5 was of such feeble vitality that it was discarded.

The chronic character of *M. melitensis* in infections in laboratory animals is a feature which renders experimental work on these organisms exceedingly slow, and it was decided to, if possible, raise the virulence of one or more strains to the value of virulence referred to by Dr. Eyre in the Reports of the Mediterranean Fever Commission (vol. ii. p. 72), so that acute infections, rather than chronic, might be studied, as the time at my disposal was brief.

The initial virulence of each of the four remaining strains was tested in the following manner:—

An agar slope of *M. melitensis* incubated at 37° C. for 24 hours, was emulsified in 0·3 c.c. saline solution and $\frac{1}{10}$ c.c. of this emulsion was injected into the brain substance of a small guinea-pig after trephining. The animal after recovering from the anæsthetic was carefully watched till death occurred, the temperature and weight being recorded daily during the period of observation.

Strains 1, 2, 3 were of a very low virulence, and the result of this experiment was as follows:—

Strain No. 1. Guinea-pig No. 107, died in 7 days.
Strain No. 2. Guinea-pig No. 108, was killed after 5 days.
Strain No. 3. Guinea-pig No. 109, died in 5 days.
Strain No. 4. Guinea-pig No. 110, died in 24 hours.

In the P.M. examination, *M. melitensis* was recovered from each animal from the blood and spleen.

Strains Nos. 3 and 4 were selected for further experiments, and an attempt was made to raise the virulence of these strains to the "Standard" (*vide ante*), that is to say of such virulence that one milligram of the culture should cause the death of the experimental animal within 48 hours from Melitensis Septicæmia.

Twice, however, the experiments had to be completely suspended owing to my illness, and to the lack of time at my disposal must be attributed the failure to raise the virulence of the strain employed to the desired point, and the work was proceeded with by means of strains of medium virulence.

Examinations made during the experiments confirm the statement that infection of the urinary tract is invariably of late appearance in *M. melitensis* infections, as in none of the experimental animals could evidence of infection be demonstrated from the bladder or urine.

The next point investigated was the effect of the injection of Di-ony-amido-arseno-benzol, commonly known as 606, upon *M. melitensis* in the tissues of infected animals. The method employed was to inoculate a series of animals with identical doses of *M. melitensis* culture, and a few minutes later to inject a dose of 606 either subcutaneously or into a vein, and observe the course of the infection.

Illustrative Series of Experiments.

In the first experiment, two guinea-pigs, Nos. 124 and 125, of similar size and weight, were taken, and an emulsion made in saline of a 48-hour old subculture from the strain No. 3. After two passages through the guinea-pig of this emulsion, $\frac{1}{10}$ c.c. $= \frac{1}{50}$th of the culture was injected intracranially, after trephining, into each of the guinea-pigs Nos. 124 and 125, while in the case of No. 125 a subcutaneous injection of 0·02 grams of 606 into abdominal wall was also given. This dose of 606 proved fatal to the animal, which died on the 4th day after the injection, and at the P.M. the drug was found to be partially undissolved at the site of the injection, with considerable necrosis of the surrounding tissues, and the heart was full of blood-clot.

The second animal, No. 124, died 11 days after injection of *M. melitensis*, having lost 20 grams in body weight, and the organism was recovered from the body tissues. Subcultures were made from these, which after 48 hours' incubation at 37° C. showed all the morphological and staining characteristics of *M. melitensis.*

In the next experiment, instead of guinea-pigs, three small rabbits were used as experimental animals. Nos. 129, 131, and 133. As before, an emulsion was made of the before-mentioned No. 3 strain. In each case 0·33 c.c. of emulsion ($\frac{1}{3}$ of a culture) was injected intracranially into the brain substance of the rabbit, after trephining the animal's skull.

In the case of rabbit No. 133, an intravenous injection of 0·013 grams of 606 into one of the veins in the animal's ear, was also given (0·013 grams 606 being equal to 0·02 grams 606 per kilo of animal's body weight).

The results of the experiment were as follows :—

Rabbit No. 132, died under the anæsthetic.

Rabbit No. 129 (Control), died after 9 days, having lost 450 grams in weight, and its temperature having steadily fallen to below 95° F.

Cultures were made from its bladder, spleen, heart, blood, and brain, and in each case showed growth of *M. melitensis.*

Rabbit No. 133, recovered from anæsthetic, and was killed with chloroform vapour 48 days after the injection. when its temperature was 103·6 F., and it had increased 760 grams in body weight.

Cultures were made from its bladder, spleen, heart, blood, and brain, which after incubation at 37° C. for 7 days showed no signs of growth of *M. melitensis.*

The next experiment was a repetition of the last, but with smaller doses of 606. Four very large rabbits were selected, and each given an intracranial injection of *M. melitensis* strain No. 4 (after passage through the guinea-pig), after 48 hours' incubation, the method being similar to the last experiment.

In addition, an intravenous (posterior auricular vein) injection of 606 was given to Nos. 137, 138, and 139 in the following doses:—

Rabbit No. 137, received 0·02 grams 606=0·008 grams 606 per kilometre of the animal's body weight.

Rabbit No. 138, received 0·02 grams 606=0·008 grams 606 per kilo of the animal's body weight.

Rabbit No. 139, received 0 07 grams 606=0·005 grams per kilo of the animal's body weight.

Rabbit No. 138, died after 24 hours from injury to its Lateral Sinus caused by the intracranial injection.

The results obtained after each animal had been carefully observed, and its weight and temperature recorded daily, were as follows :—

Rabbit No. 137, after 11 days showed 600 grams loss of weight, and its temperature was 101°·8 F., so it was given another intravenous injection of 0·04 grams of 606, being equal to 0·015 grams 606 per kilo of its body weight, with result that it showed distinct improvement, having regained 340 grams in weight when it was killed 37 days after its inoculation.

Rabbit No. 139 was killed after 40 days without having any further injection of 606. At death it showed a loss of 520 grams body weight, and also a paralysis of the right side of its neck, corresponding to the site of the brain puncture. However, on examination, there were no signs of any brain lesion.

Cultures were made from bladder, spleen, heart, blood, and brain, but after 7 days' incubation at 37° C. they showed no signs of growth of *M. melitensis.*

Rabbit No. 140 (Control), had no 606, and when killed after 37 days showed a loss of weight of 100 grams, and as in the case of 139, a paralysis of the right side of the neck.

Cultures were made from the bladder, spleen, heart, blood, and brain, and after incubation a growth which showed the morphological and staining properties of *M. melitensis* was obtained from the brain and spleen.

This completed the experimental work, owing to lack of further time at my disposal, but it is of interest to note that the late infection of the Urogenital system confirms Dr. J. Eyre's observations on that point (Report Med. Fever Commission, part 6).

Conclusions.

That a few passages of the *melitensis* through the guinea-pig are insufficient to naturally increase the virulence for that animal.

That the slightly increased virulence obtained by a few passages of the *M. melitensis* through guinea-pigs, does not increase the virulence of the strain for rabbits.

That the injection of 606 appears to have a definite inhibitory effect on *M. melitensis,* when injected into the experimental animal, shortly after the inoculation of the organism (133, 137, and 139).

That when the original dose of 606 is insufficient to prevent infection a subsequent dose appears to have a definite curative effect (137).

VI.—CLINICAL MEDICINE.

Ankylostomiasis in the North Nyasa District of Nyasaland.

By P. C. CONRAN, M.R.C.S., L.R.C.P.,
Lately Medical Officer at Karonga.

In June 1912, on my assumption of duty as Medical Officer at Karonga, I was requested by the Principal Medical Officer to investigate the extent of Ankylostomiasis infection in the district. In the course of my observations I found that a large number of cases were complicated by the presence of other Helminths in the intestines, notably Schistosomum, which are, therefore, also mentioned in my report.

It is noteworthy that my observations extended over the dry period of the year only, that is to say the period during which the minimum number of infections occur.

The arrival of a medical officer becoming known, a large number of natives came to the dispensary for treatment, not only from Karonga itself but from far distant villages.

A large number of these patients complained of pain in the epigastrium: "as if something were biting them," accompanied by constipation or, more frequently, diarrhœa, often with blood in the motion, examination of which almost invariably revealed the presence of ova of Ankylostomum, Schistosomum, or both.

The number of these cases of natives who came for treatment showed that there must be a very much greater number who, owing to the absence of severe symptoms or other reasons, did not come.

I therefore proceeded to make preparations from the stools of 522 natives who were taken, haphazard and irrespective of age or sex, from Karonga and the adjoining villages.

These preparations were examined after as short an interval as was possible, and the presence or absence of ova noted.

The results of my observations are seen in Tables I. to IV.

Table I. shows the number of persons infected with Ankylostomum, Schistosomum, Ascaris, and Tricocephalus respectively, each number being expressed both in the actual figures and as a percentage of the whole number examined.

The Schistosomum ova were in nearly every case of the lateral-spined variety: in a few cases, however, terminal-spined ova were seen, possibly owing to admixture of urine with the stool.

Table II. shows the comparative incidence of the infection in the case of men, women, boys, girls, and infants.

The results of the extent of the disease among the latter is seen in the exceedingly high infant mortality in the district, diseases from which a healthy child would easily recover proving fatal owing to lack of resisting power.

Table III. shows the variation observed with regard to locality in some of the larger villages of the neighbourhood.

It is noteworthy that Mwangolero and Mwanbungo, the villages with the highest percentage of Ankylostoma infections, lie inland at some distance from the shore of the lake, while Chirindiri and Kambombo are near the mouth of the River Rukurn.

In the two former the whole population derives its water-supply from the upper Rukurn and its tributaries; in the two latter from the Lake or wide mouth of the river.

As regards race, the bulk of the population round Karonga is composed of Wankonde. Chirindiri, however, is a Watenya village.

Table IV. indicates the relative number of pure and of mixed infections in the case of Ankylostomum and Schistosomum.

Judging from my observations Melæna is never observable with the naked eye in the case of a pure Ankylostoma infection. When Schistosomum is present, however, the case may closely simulate one of dysentery, almost pure blood being sometimes passed with marked griping and tenesmus.

Having by this time ascertained, to some extent, the condition of the Karonga district, which probably typifies the whole strip of more or less flat country lying between the Lake and the inland hills, I proceeded to Fort Hill, which is situated at an elevation of 4400 feet and is about 40 miles from the Lake.

Here I examined the stools of 100 natives from the small and scattered mountain villages. My results are shown in Tables V. and VI.

These natives were mostly of the Mambui tribe, and the water-supply is derived from mud-holes, shallow pools, and from streams which are rapidly flowing during the rains, but become stagnant during the dry season, at the latter end of which they are represented by a series of pools.

Early in December I was transferred to another station, but the foregoing observations are, I venture to think, sufficient to indicate with some degree of accuracy the state of North Nyasa in general with regard to infection with the Helminths considered.

Bass states that, in examining stools for Ankylostoma ova, some 20 cases per cent. examined are mixed. This being so, the true number of natives infected in the Karonga district is probably 60 per cent. of the population, and at Fort Hill about 33 per cent.

A few observations on the subject of the chief symptoms and signs noticed in my cases of Ankylostomum, and also on the chief difficulties to be overcome in the institution of efficient prophylaxis, may be of interest.

As regards *symptoms*, most of those usually described were observed, but certain of them were particularly emphasised.

Of these the most constant were pain and tenderness in the epigastric region, symptoms of which all the cases who came for treatment complained.

Next in frequency came dyspnœa, then palpitation, weakness, dizziness, headache. Joint pains simulating rheumatism were common.

Many patients complained of blood passed with the motions, but this was probably invariably attributable to the presence of Schistosomum infection.

From information indirectly obtained it seems certain that earth-eating is common, especially among the children. Every patient, however, stoutly denied the fact when questioned concerning his own case.

The chief signs were a dull, listless, vacant expression ; a coated, flabby, enlarged tongue, rapid, low-tension pulse, hæmic murmurs, and flabby muscles.

No reliable information concerning ground-itch could be obtained owing to the fact that my observations were made during the dry season.

Few cases had any recollection of a definite attack.

Treatment, whether with Thymol, β-Naphthol, or Eucalyptus and Castor Oil, was most successful, especially when the vermifuge was followed by a short course of iron.

In every case the patient at once began to put on weight, his intellect became clearer, the troublesome symptoms disappeared, and his capacity for work increased daily.

Prophylaxis offers difficulties which cannot be appreciated unless one is already acquainted, in some measure, with the special local conditions in North Nyasa, so that the following short description of these may be of use.

The natives belong to various tribes of which the chief are the Wankonda and the Watenga, who inhabit the strip of low-lying land along the shore of Lake Nyasa and a large portion of the mountainous hinterland as well.

Several other tribes are scattered among the hills to the North-West, such as the Mantou and Anemba, and, with a few Swahili and Arabs, complete the coloured population.

As regards their mode of living, for the present purpose the above-named tribes may be classed together.

All live in villages which, for the most part, consist of widely-scattered groups of huts, each group being connected with the rest of the village by narrow bush-paths. Each village thus covers a large area and has no definite boundaries, and, although in each case there is a head-man, his authority usually only extends over the immediate neighbourhood of his own hut.

The Wankonda and, to a lesser extent, the other tribes, are enthusiastic cattle breeders ; and all grow maize, millet, and other grain, almost the entire population working in the fields during the months immediately preceding and during the rains.

Their diet is mainly vegetarian, but fowls, eggs, and fish are used as relish, the latter being caught for the most part during the rains. It is of interest to note that Looss states that a pure vegetable diet produces a less favourable medium for Ankylostomum ova than a mixed diet. Now both fish and fowls are more abundant near the Lake, where Ankylostomiasis is rife, than in the hills, where it is comparatively rare.

Their water-supply is derived to a very small extent from the Lake ; more often from the rivers and their tributaries, especially during the rains ; most often, at any rate during the dry season, from muddy water-holes.

Defæcation and micturition are performed, whenever the desire arises, at the nearest spot sheltered or partly sheltered from the public view.

If the person concerned is on a path, he steps into the bush on one side of it ; if in a village, he makes his way to the nearest clump of bushes, a shady spot that has probably been used for years for the same purpose, and which, every rainy season, must be saturated with very slightly diluted fæces mixed with sandy soil.

During the night he is afraid to venture so far from his hut, and merely goes a few yards from his door to a spot where his children may be playing about next day.

The head-men make no attempt to set apart certain spots for latrines.

nor would such an attempt succeed owing to the scattered nature of the villages.

In these clumps of bushes near a village, therefore, we have, during the rains, all the chief factors favourable to the existence of Ankylostomum infection in a district, namely a high temperature, the optimum being from 77° to 95° F., moisture, shade, and dilution of the fæces with sand. So that during at least five months in the year, natural conditions and the habits of the people continue to produce an ideal state of things for the production of Ankylostomiasis on a vast scale.

In the dry cool months from May to September, on the other hand, and even in the heat of October and November, the conditions are much less favourable to the spread of the disease.

The South-West wind dries up the soil ; the temperature falls below the optimum ; the long grass withers, and the leaves fall from the trees, allowing the direct sunlight to reach the ground.

There are now no rains to scatter the fæces, mix them with sand, and help to apply the infective material to the skin.

In addition, the soil over a great part of the district is very efficiently disinfected ; for the natives, in order to prepare it for cultivation, kindle the long grass, and great bush fires sweep over the district, sterilising the surface of the soil.

These fires are allowed to burn close up to the huts and, by the end of November, a comparatively small area of the bush is left untouched.

Thus one would expect that there would be an annual rise in the number of fresh and of acute cases of Ankylostomiasis during the rain, followed by a fall during the dry season, and this is fully confirmed by my observations.

Any patient will date his troubles from a rainy season, and, although no reliable statistics are obtainable, the constant sound of mourning throughout the district testifies to the great rise in the mortality at this time of the year.

TABLE I.

Ova.	Number of infected persons.	Percentage of total number examined.
Ankylostomum ..	211	40·42
Schistosomum ..	169	32·38
Ascaris	47	9
Tricocephalus ..	8	1·54
Negative	87	16·66

TABLE II.

Ova.	Men.	Women.	Boys.	Girls.	Infants.
Ankylostomum . .	44	42·67	30	32·35	45·83
Schistosomum . .	31	16	48·57	44·12	29·17
Ascaris	5	11·33	1·43	8·82	22·92
Tricocephalus . .	1	1·33	1·43	2·94	2·08

TABLE III.

Ova.	Chirindiri.	Kambombo.	Mwangolera.	Mwanbungo.
Ankylostomum . .	41·6 %	25·4 %	43·4 %	50·9 %
Schistosomum . .	31·6 %	38·1 %	38·5 %	38·18 %
Ascaris	3·3 %	6·3 %	13·2 %	10·9 %
Tricocephalus . .	. . . .	. . . .	2·4 %	

TABLE IV.

Ova.	Number of infected persons.	Percentage of total number examined.
Ankylostomum alone	149	28·54
Ankylostomum + Schistosomum .	62	11·85

TABLE V.

Ova.	Number of persons infected out of 100 examined
Ankylostomum	13
Schistosomum	11
Ascaris	7
Tricocephalus	2
Negative	67

TABLE VI.

Ova.	Men.	Women.	Boys.	Girls.
Ankylostomum	15·79 %	16·22 %	12 %	5·26 %
Schistosomum	10·53 %	8·11 %	8 %	21·03 %
Ascaris	5·26 %	5·41 %	8 %	10·53 %
Tricocephalus	..	..	4 %	5·26 %

REVIEWS.

TUBERCULINS. (Notes on their Preparation and Use.)

Under the above title a brief epitome of present day practice with reference to the Tuberculins has recently been issued by the Wellcome Physiological Research Laboratories. The very copious discussion that circled round the methods of treatment initiated by Koch in 1890 at every phase of their development has accumulated in almost overwhelming mass and requires, more than most departments of medical study, the discerning hand of the sifter. The natural complexity of the subject has been intensified by the somewhat awkward nomenclature which has grown up to describe the series of substances of tubercular origin which have been employed both for treatment and diagnosis.

In this little book the various preparations are clearly defined and concisely described. The standardization of tuberculins by Koch's method and by complement deviation is described in some detail. The method of preparing New Tuberculin (W) is detailed, and it is pointed out that the dose of this preparation is stated in milligrams of bacterial substance actually present in the finished product, 2 mgm. of New Tuberculin (W) corresponding to 1 c.c. of New Tuberculin T. R. prepared from 10 mgm. of bacilli.

The various methods suggested for the employment of tuberculin as a diagnostic agent, subcutaneous, cutaneous, percutaneous, and ophthalmic, are described. This part of the subject is illustrated by coloured drawings. Calmette's ophthalmic reaction is illustrated, the colouring of the eye in a well-marked reaction of moderate severity being shown with remarkable accuracy. Tuberculin as a therapeutic agent, the opsonic index and the method of posology known as the intensive system are the subjects of brief but illuminating paragraphs.

A handy list of indications and contra-indications for tuberculin, such as a practitioner may desire to keep by him as a refresher to the memory, and a list of tuberculins available, complete this very handy and serviceable little monograph.

Tropical Medicine. By CASTELLANI & CHALMERS. 2nd edition. London: Ballière, Tindall & Cox.

Messrs. Ballière, Tindall & Cox have recently brought out a second edition of Castellani & Chalmers's 'Tropical Medicine.'

The short time elapsing between the first and second editions shows that the work has met with hearty approval and fulfils a most useful purpose.

The new edition has been brought thoroughly up-to-date, and revision, where necessary, has been most carefully carried out. We especially note with pleasure the amount of trouble that has been taken on the sections dealing with Trypanosomiasis, Kala-azar, and Filiariasis. All the more recent investigations on these diseases to which so much attention has recently been paid are described, and their value discussed. A great deal of valuable information on Fungi and skin affections in the Tropics is included, based on the authors' own work and experience.

Some 250 new illustrations have been introduced and the text has been increased by the addition of some 500 pages, but in spite of this large addition the price of the book (21/-) remains the same.

This volume is, without doubt, one of the most comprehensive and lucid manuals on Tropical diseases extant in the English language, and the authors are to be heartily congratulated on the success of the work they have produced.

H. B. N.

Diagnosis of Bacteria and Blood Parasites. By E. P. MINETT, M.D., D.P.H. 2nd edition. London: Ballière, Tindall & Cox. Price 2/6.

This little book, which now appears revised and amplified as a second edition, is heartily welcomed.

A vast amount of information is contained within its 80 pages, and we note with pleasure that the author in his additions has included and described various details of blood work which were not included in the first edition.

The little manual is not intended as a comprehensive manual on bacteria and animal parasites, but rather as a handy Vade-mecum to which the worker on the subject can readily turn when in doubt or difficulty, with the sure knowledge of finding therein just those salient points, lucidly and concisely put, which will enable him to identify with accuracy the particular organism he may be interested in.

It is a book to keep always at hand in a Pathological laboratory, and which will help the worker to overcome many difficulties.

Dr. Minett has produced a little work of great utility, and we trust in the near future to see an even more pretentious work from his facile pen.

R. O. S.

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BIBLIOGRAPHY OF DRACONTIASIS

BY

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AND
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Extract : INDIAN MEDICAL GAZETTE, July 1910.

TREATMENT OF FILARIA MEDINENSIS.

* * * * "For subcutaneous injections one drachm of a one per cent. solution (Chinosol) is used, but for a gargle or a spray 1–4 grains in an ounce of water diluted with an equal quantity of warm water is usually employed. When used in connection with this parasite, and an equal quantity of this one per cent. solution is injected on all four sides of the swelling, the total quantity used is one drachm, the object being to bathe the worm in this fluid and by so doing kill it. * * * * A long injection-needle is introduced as near as possible to the swelling, and kept about ½ inch below the skin, parallel with and along the whole length of the swelling. The needle is now slowly withdrawn, and in doing so 15 mins. of this one per cent. solution is evenly distributed along its tract. When the point of it has nearly been withdrawn it is then swung round and introduced at right angles to the first line of injection, when another 15 mins. are injected on its withdrawal. This process is repeated on the opposite side, so that the injection area forms a square and includes the whole of the guinea-worm swelling. When no suppuration was present, uniform results were obtained by this method; if the guinea-worm had not come to the surface a single injection of one drachm given in the above fashion caused its immediate death, and in four or five days the whole swelling had disappeared owing to the fact that the worm has been absorbed like a piece of aseptic catgut. On the second day after the injection the man was usually fit for his ordinary work. If the worm, however, had reached the surface but no sepsis was present the injection into the tissues killed the worm and it would safely be wound out the next day, and if by chance it broke during this process no harm was done, the worm being absorbed *in situ* and the small superficial wound rapidly healed in a few days. If suppuration was present and the case seen early, the injection killed the worm if not already dead, and markedly influenced the suppurative process, so that the healing of the wound anticipated in eight to twelve days.

"In all, 19 cases have now been treated by this method and in only one case did failure occur, and that was owing to the fact that it was not recognised that another worm was lying some three inches apart from the one which had come to the surface. The later was rapidly cured, whilst the former gave rise to an abscess gastrocnemius muscle before it was detected. Otherwise in every case treated by Chinosol injection the filaria has been killed outright and the time occupied in the treatment is at the most a fourth of that occupied by the first-mentioned method, whilst its certainty and its easier asepsis commends it as being far superior to that which Surgeon Emily advocated."

H. W. A., Lieut. I.M.S.

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